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THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 77 JANUARY, 1957 NO. 1

THE PRESENT STATUS OF PHOTOTIMING*

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PHOTOTIMING is employed universally for chest microfilming and extensively for the so-called spot filming of the alimentary tract, but except in our own laboratory, it is used almost not at all for the filming of the skull, chest, trunk and extremities—procedures which, for convenience, are designated "general roentgenography."

From the beginning, photofluorographic apparatus has been designed specifically for phototiming, and most modern spot filming fluoroscopes are designed with phototiming in mind. This suitability of apparatus design is an important factor in the success and wide acceptance of phototiming for these two phases of diagnostic roentgenology. In general roentgenography the reverse is true. Phototiming is one more accessory added to the many others that clutter most commercial apparatus, and the method has found little favor.

In our laboratory we work with apparatus designed and constructed in our own experimental shops where, unlike commercial manufacturers, we have only ourselves to please and are unhampered by the whims of customers or the claims of competitors. Our general exposing tables do not tilt and cannot function as fluoroscopes. A rigid tube arm supports the tube at a fixed distance above the center of the

CHEST	Current	Stop	CHEST	Current	Stop
C1	C	L	C4	B	S
C2	B	L	C5	B	S
C3	B	S	C6	A	S
TABLE	Current	Stop	TABLE	Current	Stop
T1	D	S	T7	F	M
T2	D	S	T8	E	M
T3	D	S	T9	E	M
T4	E	S			
T5	E	M			
T6	F	L			

CHEST	TABLE
A 50 Ma	D 25 Ma
B 100 Ma	E 75 Ma
C 200 Ma	F 100 Ma

PLATE 1. By reference to this chart, the technician can find the tube current and diaphragm stop called for by each of the fifteen phototimer settings.

* Presented at the 8th International Congress of Radiology, July 27, 1956, National Auditorium, Mexico City.

Potter grid and a 5 by 5 inch phototimer detector is permanently mounted beneath the grid. Skull roentgenography is done on similar apparatus specifically designed for that purpose, and the fluoroscopy and filming of the intestine are carried out on a special horizontal filming fluoroscope. The tube stand, cassette changer and detector used for chest roentgenography are permanently aligned for that work and are not employed for other purposes.

This combination of specificity and simplicity of our apparatus antedated phototiming and is important in its own right, but it must be admitted that it facilitates phototiming. All commerical manufacturers have engineers who are capable of designing clinical roentgen-ray apparatus that is as precise and rugged, as, for example, good quality three-drawer correspondence files, milling machines, or precision lathes, but their work is hampered by the conviction that every piece of diagnostic roentgen-ray equipment must be capable of performing almost every type of roentgen examination. Instead of designing a table that will do the best possible job of filming of the trunk and extremities, engineers are required to incorporate a tilting fluoroscope as well. On the other hand, spot filming fluoroscopes must be loaded down with accessories to enable them to function also as general filming tables. The Potter grid (so-called Bucky

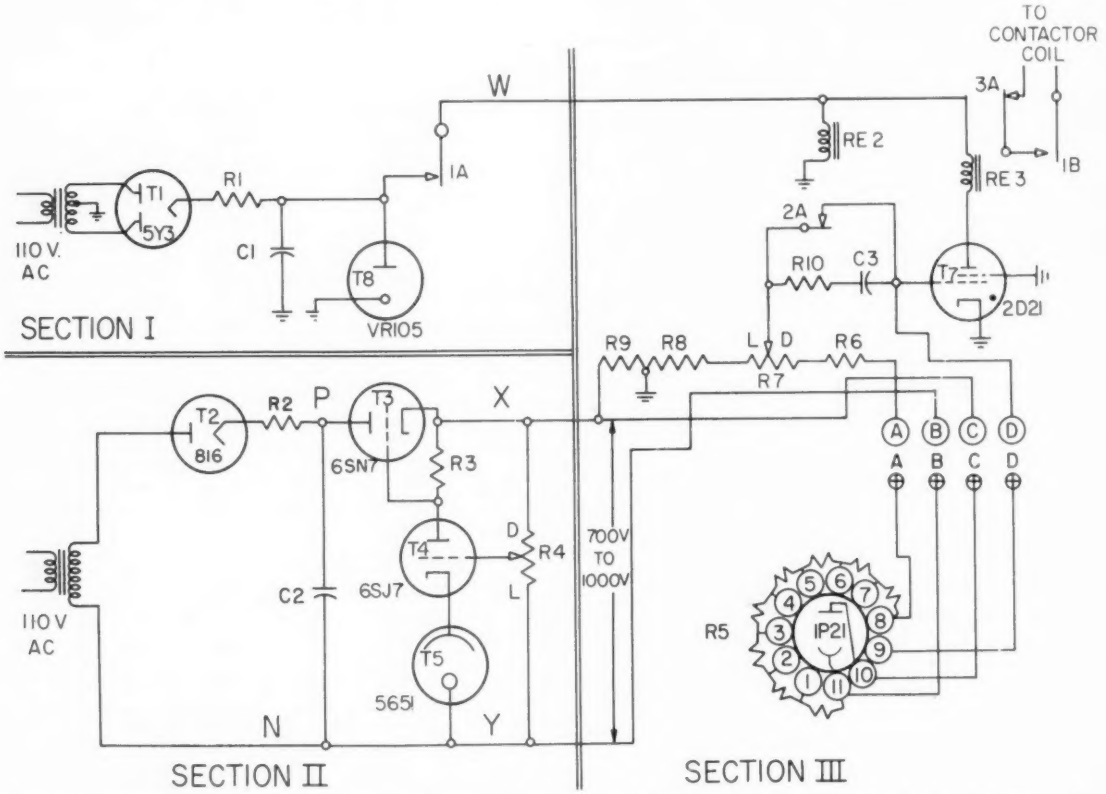

FIG. 1. *Schematic Diagram of Phototimer Circuit.* Published originally in 1950[2] and described in detail in that publication. Originally T_3 was a 6J5 but more recently we have substituted a 6SN7, which is able to carry heavier currents. Section I is the low voltage D.C. supply, Section II the high voltage D.C. supply, and Section III the detector and trigger tube assembly. R_7 establishes the initial bias on the grid of the thyratron (T_7) and thus regulates the sensitivity of the phototimer. When a roentgen ray exposure is being made, the cathode of T_6 (electrode 11) receives light from a detector screen and a photo-current flows from the cathode to dynode after dynode and eventually to the anode (electrode 10). This current flow draws electrons from C_3 to dynode 9 and when the bias on the grid is reduced to -3 volts T_7 fires, terminating the roentgen ray exposure.

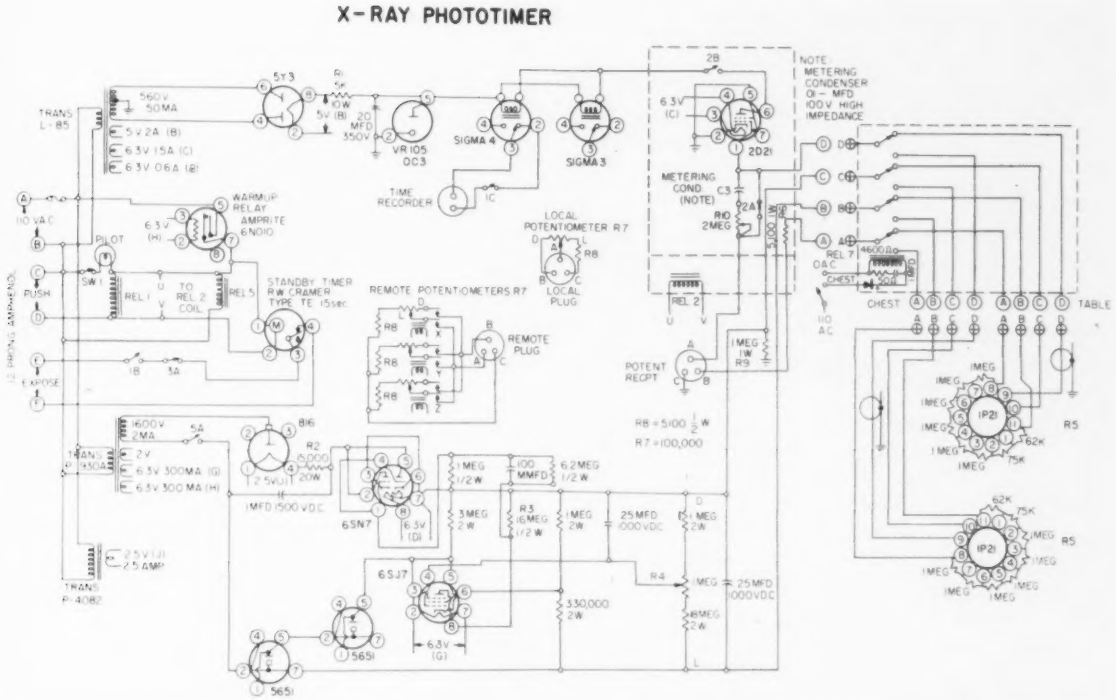

FIG. 2. *Actual Wiring Diagram of Phototimer.* The principal changes since the 1950 publication are: (1) The 816 is subjected to high voltage only after a delay to allow for the heating of its cathode. This is accomplished by relay 5 (Guardian type 110 A.C.). It has one set of N.O. contacts (5_A) carried on ceramic mounts. When the Amprite 6NO10 (filament 10-second time delay) closes, contacts 5_A close, applying high voltage to the 816. (Relay 5 formerly was a 110-volt D.C. relay with single N.C. contacts that opened when the stand-by timer called for termination of the roentgen-ray exposure. No relay is required with the type T.E. Cramer timer because it has isolated contacts.); (2) The filament of 816, formerly operated at 2.0 volts A.C., now receives 2.5 volts from transformer P-4082, made by the Chicago Standard Transformer Company; (3) A 6SN7 has been substituted for the 6J5. (4) There are changes in the ohmage and wattage of several of the resistors; (5) Relay 7 has been added to distribute either of two detectors to the chassis. This is a hermetically sealed relay manufactured by the Guardian Electric Manufacturing Company of Chicago. The catalog number is 595. Operation is on 110 volts D.C. The contacts are 4 P.D.T. palladium. The contacts that distribute the signal from dynode 9 over nib D of the 4-prong amphenol connector are mounted in a separate stack to assure maximum insulation. The other three contacts are mounted in another stack.

diaphragm) must be made thin enough for incorporation in tilting fluroscopes even though doing so requires that the cassette tray be a flimsy affair, devoid of antifriction bearings and incapable of trouble-free performance in a busy laboratory.

Market surveys undoubtedly are the basis of the belief that customers prefer general purpose roentgen-ray equipment, but in other fields of manufacturing it has been demonstrated that courageous pioneering sometimes will create markets where none existed before. I believe that if some manufacturer were to offer a line of custom-built, simplified, specialized, heavy-duty roentgen-ray apparatus intended for use in large, busy radiologic departments, he would shortly build a market for such wares and, incidentally, would stimulate the more extensive use of phototiming in general roentgenography.

PHOTOTIMER DESIGN

Our phototimers consist of the following parts and subparts:

1. Chassis
   - A. Voltage regulator
   - B. Bias regulator

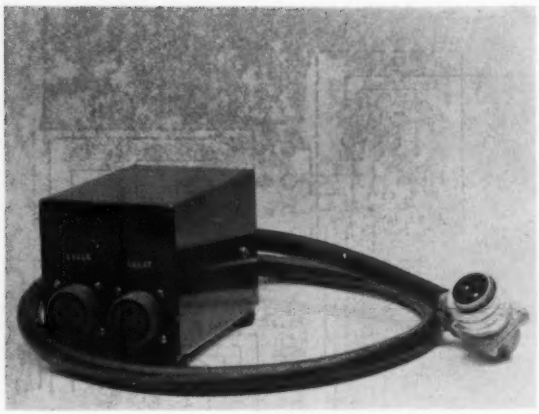

Fig. 3. *Dectector Distributor.* The housing contains relay 7 of Figure 2. It is a sealed relay (catalog no. 595, Guardian Electric Manufacturing Company of Chicago, 110 volt D.C., 4 P.D.T. palladium contacts). The cable that is attached to the distributor plugs into the chassis and the individual detector cables plug into the distributor. When any of the T settings are being used (see Fig. 5) the coil of relay 7 is not energized and its N.C. contacts connect the table detector to the chassis. When any of the C settings are employed the coil of relay 7 is activated and it is the chest detector that is connected to the chassis.

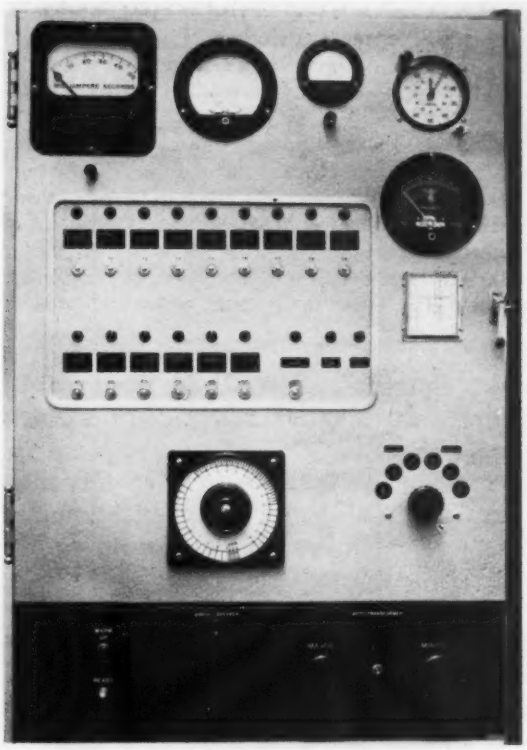

Fig. 4. *Control Panel of Roentgen-Ray Machine Equipped for either Manual or Automatic Operation.* Top row, reading left to right: Ballistic milliammeter, which is automatically in circuit for high current settings or by means of push-button switch below meter can be put in circuit for any setting; next, A.C. milliammeter; next, valve tube meter. This is a small double-throw D.C. milliammeter wired in series with the A.C. milliammeter but normally shorted out of circuit by the N.C. push-button switch that is located immediately below it. At the beginning of work each morning and afternoon, the technician makes a timed exposure while she presses the push-button to unshort the valve tube meter. If the filaments of all four valve tubes are burning, alternating current is fed to the valve tube meter and its needle vibrates about the central zero position. If one or more of the valve tubes is nonconducting, the machine operates as a half-wave set and the needle of the meter deflects strongly either clockwise or counterclockwise. Next is a time recorder, which may be cancelled by the lever shown at 11 o'clock on the dial or may be put out of circuit by a toggle switch at 5 o'clock. The time recorder is used principally during the trial and error testing that brings the various settings into calibration. Below the time recorder is the kilovolt meter and below it a chart showing the current and diaphragm stop called for by each of the 9 T settings and the 6 C settings. (See Plate 1). At the bottom row center is the dial of a Liebel-Flarsheim electronic timer calibrated in 22 steps from 0 to 1 second, in $\frac{1}{8}$ seconds from 1 to 3, in $\frac{1}{4}$ seconds from 3 to 10, and in $\frac{1}{2}$ seconds up to 14 seconds. Lower right: Dial switch for manually selecting 50 ma. (A), 100 ma. (B) or 200 ma. (C) for the chest tube; or for selecting 25 ma. (D), 75 ma. (E) or 100 ma. (F) for the table tube. In the center is the phototimer panel, which is described in the legend for Figure 5.

2. Detector
   A. Fluorescent screen
   B. Light pipe
   C. Masks
3. Detector cable
4. Detector cable distributor
5. Detector diaphragms or stops

The reader who wishes to understand our apparatus and the way we use it in general roentgenography will find the present paper more understandable if he begins by reading three earlier publications from this laboratory.[1,2,3] The space available here allows little repetition except where significant changes have been made. Figure 1 is a schematic diagram, Figure 2 an actual

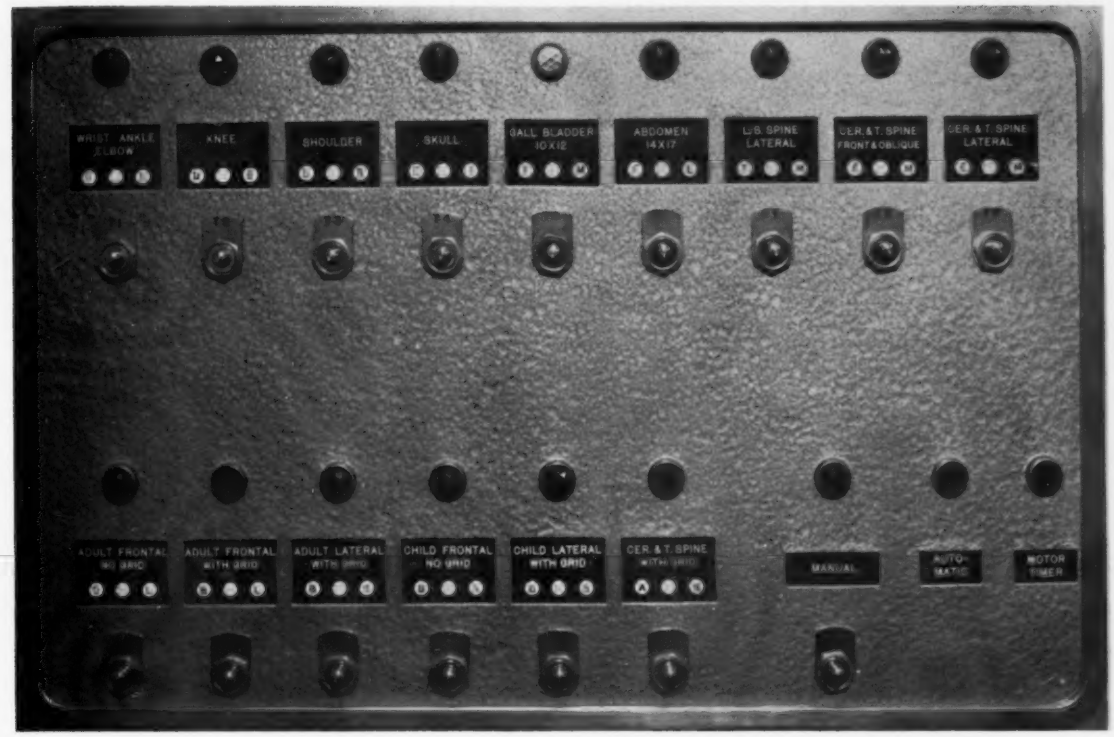

FIG. 5. *Phototimer Panel.* At the top are the 9 table settings designated T_1, T_2, etc. At the bottom left are the 6 chest settings designated C_1, C_2, etc. At the time this photograph was made, the push-button for T_5 had been closed, thus setting up the machine for making small films of the gallbladder region or the hip or the thigh. The momentary closing of the push-button for T_5 initiated the following train of events: (1) The high tension switch was thrown to the circuits for the table; (2) A tube current of 75 ma. was selected; (3) The medium-sized diaphragm stop was brought into position between the film and the detector; (4) The pilot light above T_5 was lighted; (5) The exposing switch was connected to the circuits of the phototimer; (6) An R_7 calibrating potentiometer was selected, which had previously been calibrated by trial and error to produce good gallbladder films. If one now wished to operate without the phototimer, he would press the push-button farthest to the right in the lower row. This would extinguish the light above T_5 and turn on the light above the small panel reading "Manual." Current would now be selected by manually operating the dial switch shown in Figure 4 and the exposing switch would be put in circuit with the Liebel-Flarsheim timer. The labels for each of the fifteen settings are engarved on laminated bakelite and the current setting, kilovoltage and symbol for diaphragm stop are engraved on lucite buttons that are pressed into holes drilled along the lower edge of the labels. When the control stand is energized, all fifteen of the labels are illuminated but only in the case of the setting that is being used in the pilot light lighted. In the case of setting T_5, the left-hand button reads E, indicating a current of 75 ma., the central button reads 80 because the setting has been calibrated for a kilovoltage of 80, and the right-hand button reads M, indicating that the medium-sized diaphragm stop is used. The engraving on the right-hand and left-hand buttons has been filled with black paint, which records well on the photograph, but the central button showing the kilovoltage to be used is filled with red paint and so does not reproduce well in the photograph. There is considerable practical advantage in using removal buttons for current, kilovoltage and diaphragm stop because as our ideas change and we wish to change these combinations we have merely to remove the old buttons and insert new ones, without discarding the entire label.

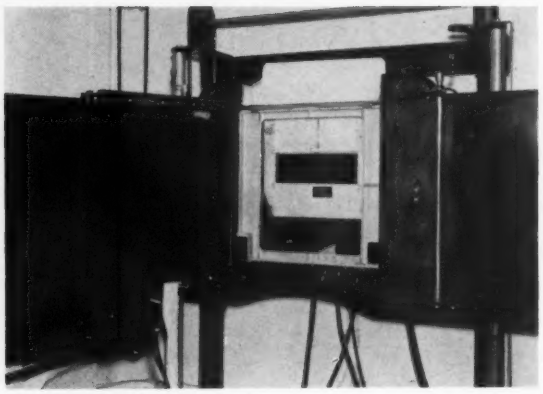

Fig. 6. *Keleket Cassette Changer for the Making of Stereoscopic 14×17 inch Chest Roentgenograms.* The detector has a $3\frac{1}{2}$ inch by 10 inch zinc cadmium sulfide screen attached to the back of a bakelite panel, which is seen in this illustration. A detector of this sort integrates a broad band of radiation coming through the upper thirds of both lungs and the upper mediastinum.

wiring diagram of the electronic circuits in the phototimer chassis. Figure 3 is a photograph of the detector distributor (relay 7 of Fig. 2). Figure 4 shows the entire control panel, and Figure 5 the phototimer portion of that panel in one of our roentgen ray machines.

Fig. 7. For children's chests or lateral chest filming in adults, the full breadth detector would integrate too much grazing radiation and so we employ a central, rectangular diaphragm stop $2\frac{1}{2}$ inches wide and 3 inches high. When setting C_1 of the phototimer is used (see Fig. 5) the full aperture of the detector is employed and the pilot will not light unless the grid is swung out of the way. With C_2 the pilot will not light unless the grid is in position, and for C_3 the grid must be in position and the small stop is brought into position automatically.

MULTIPLE SETTINGS

Our machines provide for 15 separate "settings," 9 for use on the exposing table, designated T_1, T_2, T_3, etc., up to T_9, and 6 for the chest-filming apparatus, designated C_1, C_2, C_3, etc., up to C_6, (see Fig. 5). The significance of "settings" may be explained by describing three of them. T_2 is used for the knee, T_5, for the gallbladder, thigh and hip, and T_6 for abdomen and pelvis. For filming the knee, we prefer rather low kilovoltage and low current so that the exposure time will not be so short as to produce grid lines on the film, and it is necessary that we use the small diaphragm stop so

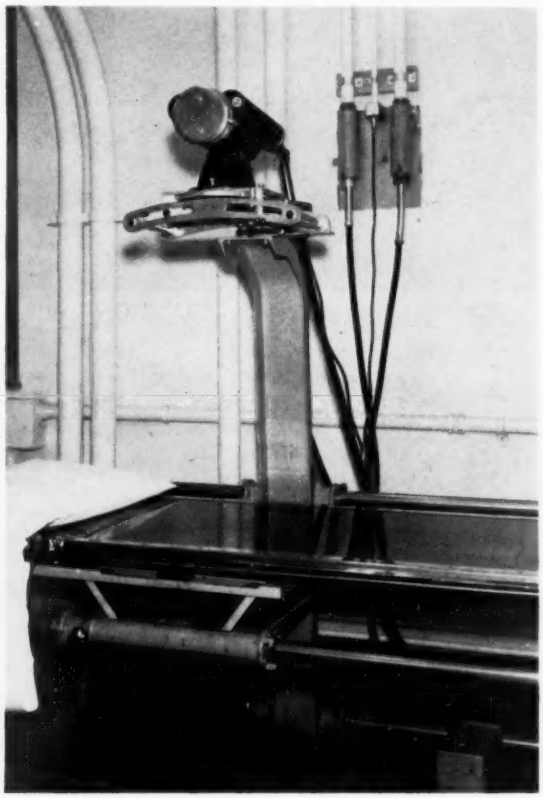

Fig. 8. *General Exposing Table Equipped for Phototiming.* A 16 to 1 Liebel-Flarsheim grid is used and a Dynamax 46 rotating anode tube is mounted 40 inches above the center of the film. The grid and tube may be moved longitudinally, the length of the table and the tube may be shifted 3° from center along an arc for stereoscoping. It is not possible, however, for the cone of radiation to fall anywhere except with its axis at the center of the film and at the center of the phototimer detector.

that the radiation read by the detector will have come through rather than around the knee. T_2, therefore, calls for 70 kv. and 25 ma., and its R_7 potentiometer has been calibrated by trial and error to give the density we desire for knees and other anatomic structures of about that size.

For gallbladder, hip and thigh, we use the medium stop, and the parts are so heavy that it is desirable to work at 80 kv. and 75 ma. in order to keep exposure time within reasonable limits. T_5, therefore, calls for 80 kv., 75 ma., and the medium stop, and its R_7 potentiometer has been calibrated for optimum density for structures of approximately the density of the upper right quadrant of the abdomen.

For 14×17 inch films of the abdomen or pelvis, it is necessary to use the large stop in order to integrate a large cross section of the radiation passing through the patient's body, and rather high kilovoltage and current are needed. T_6 calls for 80 kv., 100 ma., and the large stop, and its R_7 potentiometer is calibrated by trial and error for pelvic films of optimum density.

The kilovoltage that should be employed for each of the 15 settings is shown on the illuminated labels, but, unlike current and diaphragm stop which are selected automatically when the push-button is pressed, kilovoltage must be dialed by hand.

The fact that the roentgen tube is permanently mounted perpendicularly above the cassette tray of the grid with the phototimer detector immediately beneath the center of that tray has no disadvantages in 14×17 inch filming of the pelvis and abdomen because, entirely aside from phototiming, it is necessary for the technician to arrange the patient rather carefully if all the parts of the pelvis are to show on the film. It would require extreme carelessness to do centering so poorly as to interfere with satisfactory phototiming. The situation is quite different in the making of small films (8×10 inch or 10×12 inch films) of such parts as the gallbladder, hip, thigh, knee, ankle, shoulder, etc. Here the employment of phototiming requires that the small film be placed with its center on the axis of radiation and that the part be carefully centered on the film. For most patients it is not difficult to do this, but particularly for those who are wearing plaster casts it may be simpler to use a large film, make no effort to center the part on the film, and use mechanically timed exposures rather than phototiming. As is pointed out in the legend for Figure 5, our machines can be changed over from conventional manual control to phototiming by the mere pressing of a button. Where one has to do a large amount of roentgenographing of fingers and toes, hands and feet, using film without the grid, it is a simple matter to set up a film tunnel

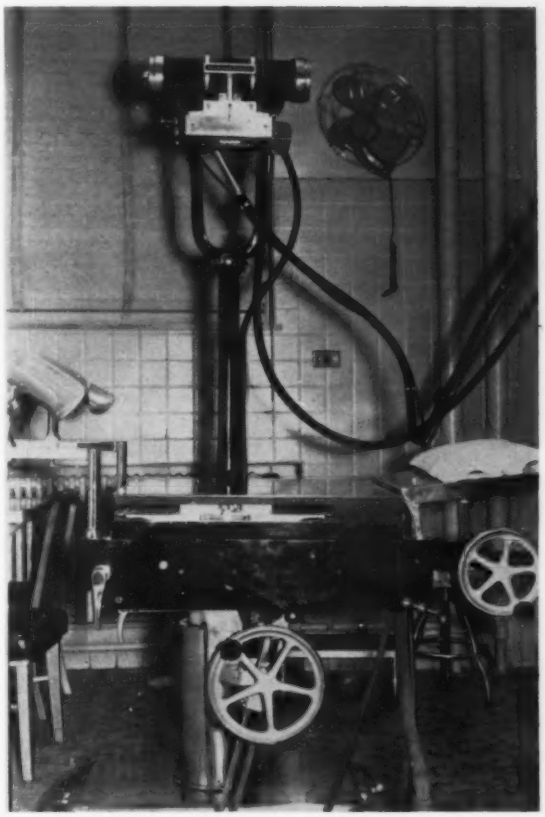

Fig. 9. *Cystoscopic Table Equipped with 16 to 1 Ratio Liebel-Flarsheim Grid, a Thin Phototimer Detector with Lucite Light Pipe, and a Rigid Tube Arm That Supports a Dynamax 20 Tube 40 Inches above the Center of the Grid.* Stereoscoping may be accomplished by displacing the tube 3° either way from center along a curved track.

equipped with a small aperture phototimer detector. It is possible also to provide special R_7 potentiometers so that such so-called "table-top raying" can be accomplished on our general roentgenographic tables by having the radiation that has passed through the part and the film continue on through the table top, through the grid into the detector that lies beneath it. Practically, however, we find it more convenient to use mechanical timing for the filming of such small parts.

In our skull apparatus, which is not pictured here, we have separate phototimer settings for 90° sinus films, 60° sinus films, occipital views of the skull, lateral views of the skull, mastoids, Stenver's views, etc.

CONCLUSION

All photofluorographs and most spot filming fluoroscopes are designed with phototimers as integral parts, but in general roentgenography if phototimers are used at all they are added as accessories to apparatus already over-encumbered by accessories.

Phototimers themselves have been perfected to the point where they will render dependable service for long periods of time, requiring little in the way of servicing. They are not apt to enjoy large-scale employment in general roentgenography, however, until more suitable roentgen-ray apparatus becomes available for that type of work.

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Chicago 37, Illinois

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VOL. 77, NO. 1

EXFOLIATIVE CYTOLOGY AS AN AID IN THE DIFFERENTIAL DIAGNOSIS OF GASTRIC LESIONS DISCOVERED ROENTGENOLOGICALLY*,†

By CYRUS E. RUBIN, M.D.,‡ *and* JAMES F. NELSON, M.D.§
SEATTLE, WASHINGTON

CELLS and tissue particles exfoliated from the gastric mucosa were first used in the diagnosis of cancer more than half a century ago.[2,5,9,10,25,39,41,42,51] With the advent of radiology and endoscopy, cytology was largely forgotten for the next five decades. The recent revival of this method has been stimulated largely by the excellent results obtained in cervical carcinoma.[28,36] The initial technical difficulties[12,17,23,35,37,40,52,54,57,58] in obtaining cells from the stomach have been surmounted. Current reports are most encouraging.[7,8,15,16,34,43,46,48,49,50,55]

Under normal circumstances epithelium is continuously shed from the surface of the gastric mucosa. The decreased mutual cohesiveness of the cells comprising a malignancy[6,29] increases the rate of exfoliation. This convenient property makes it possible to diagnose very small cancers.[21] It is thus apparent that the examination of these cells should be of practical clinical value. To test this hypothesis a four-year study was made of exfoliative cytology as a complement to the usual diagnostic work-up of suspected gastric cancer.

Experienced personnel now attain a cytologic accuracy of 80 to 90 per cent.[4,16,21,50] Forty-nine of 56 proved gastric cancers (88 per cent) studied in this laboratory were correctly diagnosed by cytologic examination prior to surgery. The procedure is only reliable if the patient has been properly prepared to ensure a clean stomach which is free of food and detritus. Furthermore, efficient methods must be used to obtain the cells before they become digested.

Various collection techniques have been proposed which use abrasion[34] or enzymes[43] to promote exfoliation. Gastric lavage with a buffered solution of the enzyme chymotrypsin is the routine method now employed in this laboratory.[47] It is simple and accurate.[21,47] In many cases, the search for diagnostic cells is not too time consuming, but occasionally, many man-hours are required to find them. Considerable experience is required for accurate diagnostic interpretation. The trained personnel and the equipment required, although considerable, are less than that needed for operating an adequate diagnostic radiology service.

Selected cases follow which presented a problem to the radiologist and clinician. These cases were especially chosen to illustrate the usefulness and limitations of the gastric cytologic method.

GASTRIC ULCERS

A definite roentgenologic decision regarding the malignant nature of a gastric ulcer is often difficult. It is seldom necessary to resort to laparotomy for diagnosis. In the majority of cases the correct preoperative diagnosis may be made by the intelligent integration of the roentgenologic, cytologic, gastroscopic and clinical findings.[48,50]

REPORT OF CASES

A well-preserved fifty-one year old man* entered the hospital because of almost continuous epigastric pain of five weeks' duration. The pain was not relieved by food and was

* Reported in detail in Reference 50.

* Presented at the Fifty-sixth Annual Meeting of the American Roentgen Ray Society, Chicago, Illinois, September 20–23, 1955.
† This project was supported by funds accruing to Initiative 171 of the State of Washington for Research in Biology and Medicine, by a Damon Runyon Research Grant, and by the Washington State Division, American Cancer Society.
‡ Department of Medicine, University of Washington, Seattle, Washington.
§ Department of Radiology, University of Washington, Seattle, Washington.

most severe at night. Severe anemia and 4 plus occult blood in the stools were present. A high lesser curvature ulcer, visualized roentgenologically, was interpreted as a probable ulcerating carcinoma. Gastroscopy was not entirely adequate; a questionable mass on the anterior wall was visualized. The cytologic examination revealed only benign cells. At laparotomy the gross characteristics confirmed the roentgen impression of cancer and a total gastrectomy was performed. On pathologic examination this proved to be a large benign ulcer surrounded by an unusually prominent rim of inflammatory edema. The patient died seven days after operation from leakage at the site of anastomosis.

From the foregoing and other similar experiences, we believe that a negative cytologic examination justifies delay for more extensive diagnostic studies. Although a negative cytologic examination does not rule out the presence of malignancy, it is of definite significance. The correct diagnosis in the case described above might have been established by a roentgenographic examination demonstrating healing after antacid therapy or by another gastroscopy with more adequate visualization. A correct preoperative diagnosis of benign gastric ulcer would have prompted more conservative treatment and might have saved the patient's life.

An obese, fifty-nine year old man entered the hospital because of continuous angina pectoris requiring opiates around the clock. The physical findings of aortic stenosis were present, and an aortic valvulotomy was contemplated. An upper gastrointestinal roentgen examination was ordered because of minimal epigastric discomfort. The roentgenogram demonstrated a high lesser curvature ulcer which was interpreted as a possible carcinoma because of the adjacent filling defect (Fig. 1). Gastroscopic visualization of the lesion was impossible because the tip of the instrument caught in the ulcer floor. Cytologic examination yielded no malignant cells and therefore a trial of antacid therapy was suggested. Shortly after institution of this therapy angina decubitus disappeared and opiates were no longer required. The ulcer took many months to heal. Eventually, radiation therapy was used as an adjunct to the medical regimen with complete healing. The patient was able to climb hills without angina pectoris, although more severe exertion would cause chest pain.

A frail, seventy-nine year old woman was hospitalized because of nausea, anorexia and 20

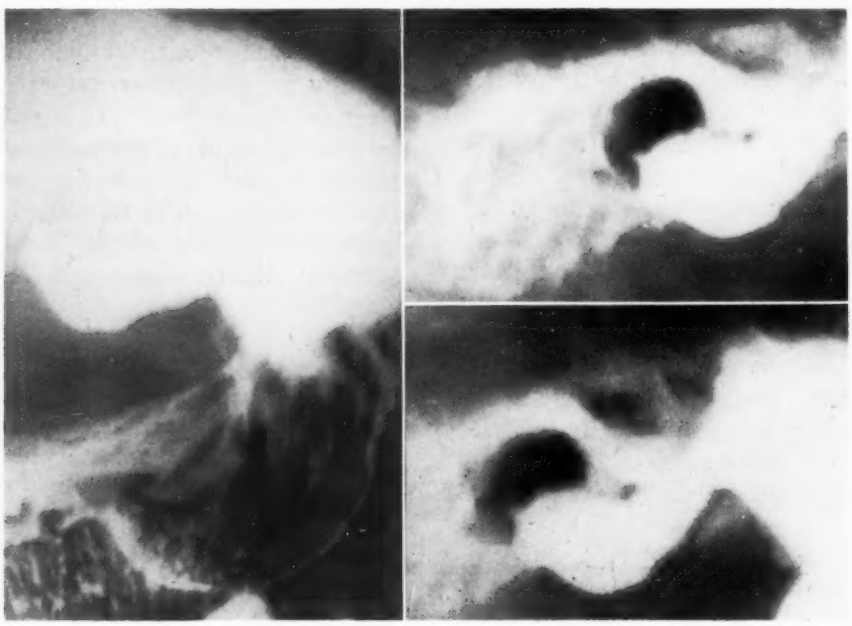

FIG. 1

Fig. 2 (*A* and *B*)

pounds weight loss over one year's period. The initial gastric roentgenogram (Fig. 2*A*) was interpreted as a high lesser curvature malignant ulcer. Cytologic examination showed no malignant cells. The patient was therefore started on medical therapy. The ulcer healed almost completely within three weeks and further roentgenograms showed complete healing (Fig. 2*B*).

A sixty-one year old man with a recent myocardial infarction entered because of hematemesis. A lesser curvature ulcer was demonstrated which didn't project entirely beyond the confines of the stomach. It was felt by the radiologist that a neoplasm could not be ruled out, though it was most probably a benign ulcer. Cytologic examination demonstrated no malignant cells. This reinforced the clinicians in their resolve to treat this patient medically. Antacid therapy produced complete healing of the ulcer. Radiation therapy was given to make recurrence less likely. Achlorhydria was present a year after institution of treatment.

Although surgery may be an effective treatment for benign, gastric ulcer, it is not always the wisest course of action in elderly patients or in those with complicating disease. If one can be assured of the benignancy of a lesion, a safer, more conservative regimen may be followed.

FUNDAL LESIONS

The difficulties in roentgenologic examination and interpretation of fundal lesions are well recognized. Moreover this area is relatively inaccessible to gastroscopic visualization. Cytology may be especially helpful.

REPORT OF CASES

A seventy-one year old Scandinavian man* had known pernicious anemia for twenty-three years. He had been carefully followed. Because of several weeks of dyspepsia, a roentgen exami-

* Reported in detail in Reference 45.

nation was ordered. A pouch-like defect of the fundus was interpreted as a variation of normal. The gastroscope would not pass the lesion; therefore, a fine polyethylene tube was introduced into the area. Characteristic large columnar cells, such as are seen in all cases of pernicious anemia, were found.[27] Other cells characteristic of adenocarcinoma were also noted. A repeat roentgen examination was then interpreted as suggestive of neoplasm of the fundus. At operation, a carcinoma of the cardia extending into the porta hepatis was found.

This case illustrates the difficulties of diagnosing fundal lesions. It also serves to emphasize the fact that gastric carcinoma occurs more frequently in pernicious anemia.[3,18,19,20,30,31,32,38,44,53,56] Curable asymptomatic cancer of the stomach may be demonstrated by annual diagnostic studies.[44,45,53] Gastric exfoliative cytology is especially useful in this type of selected screening.[13,45]

An eighty-six year old woman had six months of intermittent epigastric distress. A roentgenogram demonstrated a large hiatus hernia with a filling defect, as well as a large gastric ulcer at the level of the diaphragm (Fig. 3). The ulcer healed on a medical regimen. Cytologic examination revealed definite malignant cells, but they were rare and hard to find. Because of the S shape of the hernia, esophagoscopy and gastroscopy were impossible.

In this case cytology confirmed a roentgen suspicion in an area that was inaccessible to palpation or endoscopic visualization.

A seventy-seven year old woman entered the hospital because of melena and hematemesis of recent onset. A hiatus hernia was obvious on the roentgenogram. There was a constant filling defect in the infradiaphragmatic fundus (Fig. 4). Esophagoscopy and gastroscopy suggested a mass in the same area. The cytologic examination showed atypical cells but no definite malignancy. At operation the area in question was minutely examined after the stomach was opened. No cancer was found. The subsequent course of more than a year has confirmed the

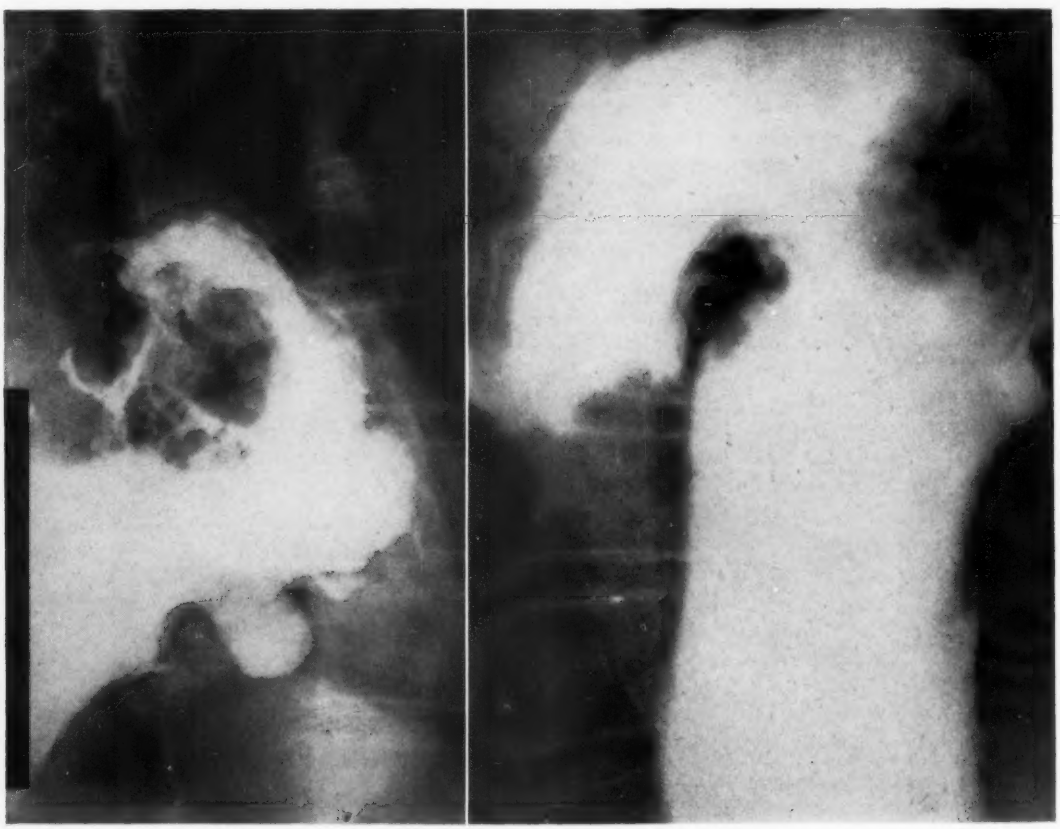

FIG. 3

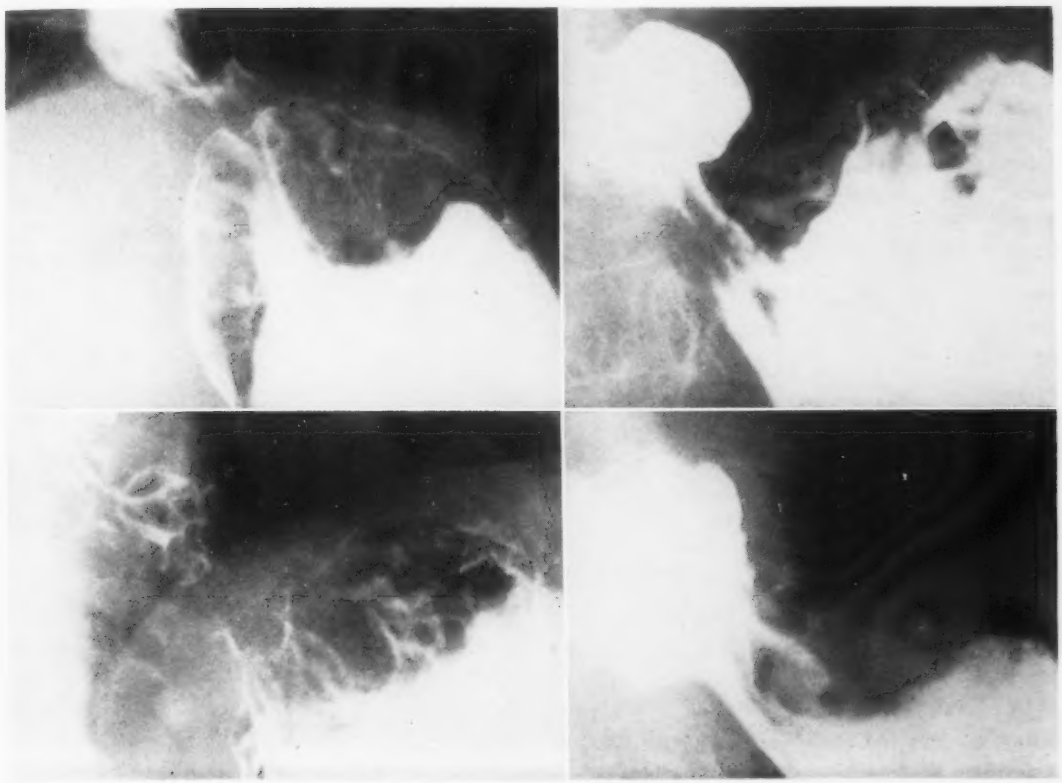

Fig. 4

impression of a benign lesion. The roentgenograms continue to have the same appearance.

In the foregoing case none of the methods enabled a definite, correct preoperative diagnosis of this confusing fundal lesion. Apparently this filling defect was an artefact secondary to a hiatus hernia.

A seventy-five year old man entered the hospital because of epigastric pain of two years' duration accompanied by marked weight loss. His roentgenograms were interpreted as a possible neoplasm of the upper third of the stomach (Fig. 5). The cytology was typical of lymphoma.[49] The patient died of exsanguinating hemorrhage before radiation therapy could be instituted. A reticulum cell sarcoma of the stomach was demonstrated at necropsy.

In this instance the cytologic method established the malignant nature of a lesion suspected roentgenologically. Furthermore, it established the lymphomatous nature of the process.

HEAVY FOLDS

Roentgenologic interpretation of heavy fold patterns may be exceedingly difficult. Gastroscopy is of some aid in differential diagnosis, but it too may be misleading. If technically feasible, gastroscopic biopsies under direct vision are quite helpful. The following cases illustrate the role of cytologic examination in these problem cases.

REPORT OF CASES

A fifty-four year old man* had intermittent epigastric pain of six months' duration. He lost 36 pounds during this period. On physical examination his liver was enlarged. The initial gastric roentgenogram was interpreted as a heavy fold pattern on the greater curvature of indeterminate etiology. A repeat roentgenogram was considered to be normal. The initial attempt at gastroscopy was unsuccessful. Cells characteristic of adenocarcinoma were obtained (Fig. 6, upper group). The cytologic criteria which enabled this diagnosis to be made were:

* Reported in detail in Reference 48.

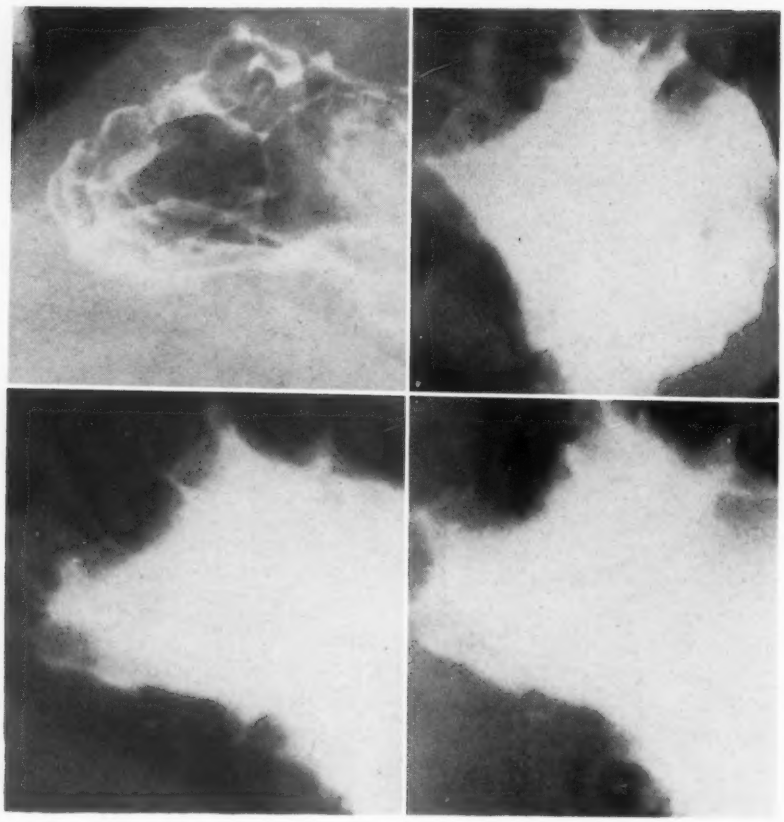

FIG. 5

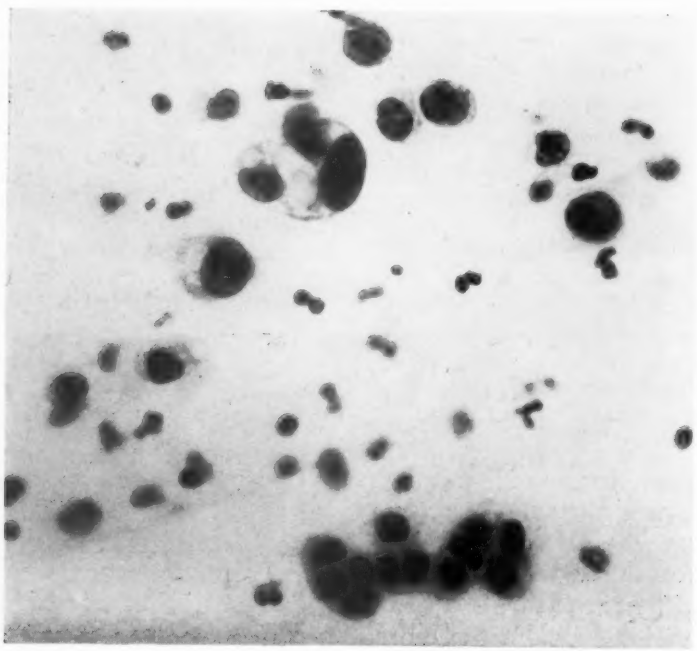

FIG. 6

variation in nuclear size, increased nuclear chromatin content and cytoplasmic vacuolation. A group of normal gastric columnar cells (Fig. 6, lower group), which exfoliated from the uninvolved stomach in this patient, may be contrasted with the malignant ones. Gastroscopy now showed infiltrated folds suggestive of carcinoma. The cytologic and gastroscopic findings resulted in prompt operative intervention. Carcinoma was proved on histologic examination.

The cytologic method established the malignant nature of the heavy folds in this patient. A number of gastric lesions of uncertain nature have been proved malignant when exfoliative cytology was integrated with other diagnostic procedures.

For twelve years this forty-four year old man had experienced recurring episodes of burning epigastric pain which were relieved by food. His gastric roentgenograms were interpreted as an ulceration within a mass representing a lesser curvature carcinoma (Fig. 7). At gastroscopy giant folds were seen, and the differential diagnosis lay between giant hypertrophic gastritis and infiltrative carcinoma. A biopsy was obtained with the Benedict gastroscope. It showed no evidence of malignancy. The cytologic interpretation was benign. At operation giant hypertrophic gastritis was found. Several tiny peptic ulcerations were located between the heavy folds which resembled cerebral convolutions.

This is an instance of that rare entity giant "pseudotumoral" hypertrophic gastritis. The confluence of folds mimicked an ulcerating carcinoma roentgenologically. The gastroscopic biopsy and exfoliative cytology pointed to the nonmalignant nature of the process.

For six months a seventy-two year old man complained of cramping abdominal pain which was relieved by food and antacids. The large mucosal folds demonstrated on the roentgenogram led the radiologist to suspect a submucosal infiltrative lesion (Fig. 8). At gastro-

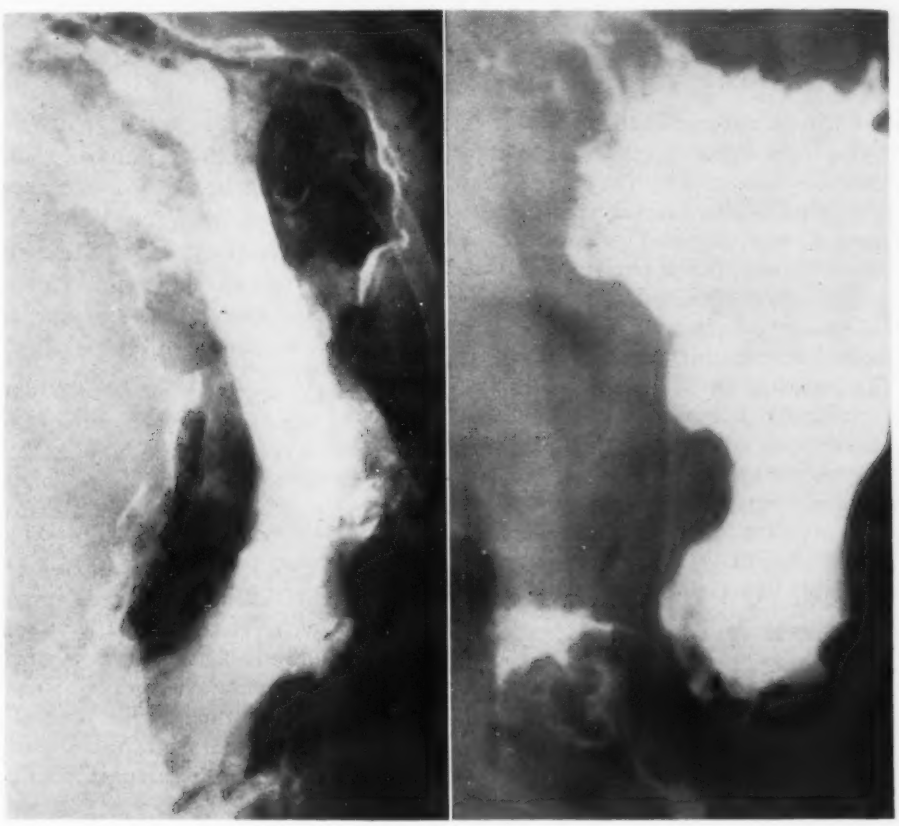

Fig. 7

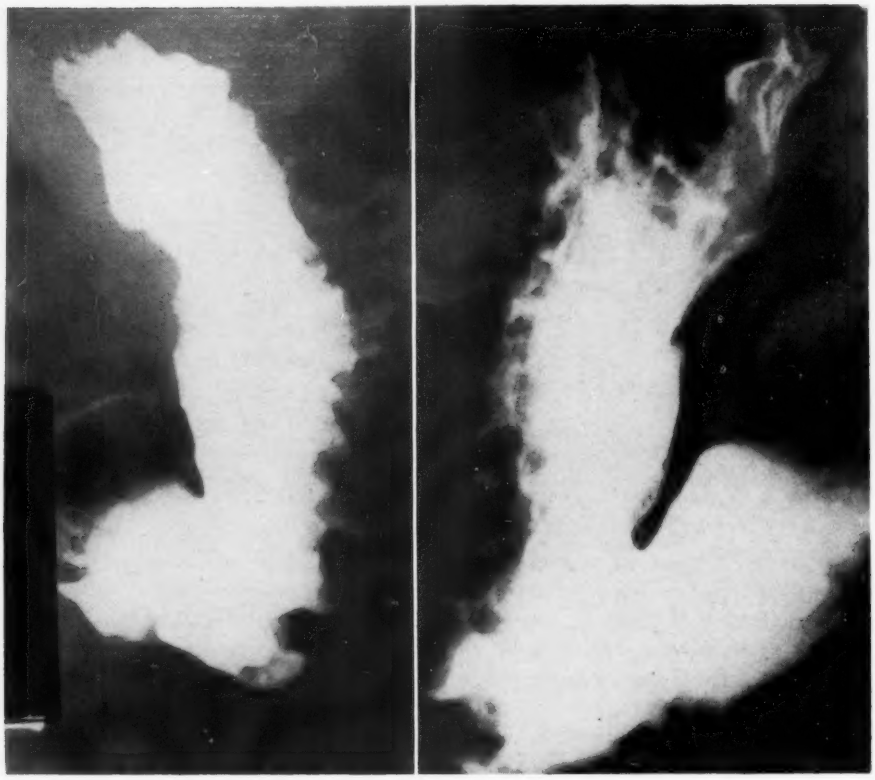

FIG. 8

scopy giant folds were seen and the possibilities of infiltrative carcinoma or giant hypertrophic gastritis were considered. Upon repeating the gastroscopy a small ulcer was visualized with a nodular area in one margin. This prompted a diagnosis of carcinoma. A few poorly preserved malignant cells were obtained. At operation gross examination of the excised stomach showed giant hypertrophic gastritis with no evidence of cancer. Later careful search of the operative specimen revealed a small ulcer hidden between two adjacent enlarged gastric folds. A tiny nodule on the ulcer's edge proved to be a carcinoma histologically. There were no lymph nodes involved. The patient has done well for more than a year since operation. This may possibly prove to be a cure.

The above case is another instance of giant hypertrophic gastritis (not to be confused with that common gastroscopic misdiagnosis, "hypertrophic gastritis," which has been proved by suction biopsy to be a variation of normal).[33] Several authors have emphasized the extreme difficulty of correctly diagnosing this rare lesion roentgenographically or by gastroscopy.[1,24] Cytology may be especially helpful. It is interesting that the possible premalignant nature of this disease has been suggested.[1] The above case may represent an instance of malignant transformation. Some of the roentgenologic difficulty in demonstrating these large folds may be explained by the thick mucus which obscures the sulci.[11]

A seventy-six year old woman entered the hospital because of three months of abdominal pain and a recent hematemesis. The roentgen examination suggested massive neoplastic involvement of the stomach. Because of the heavy fold pattern, the possibility of lymphoma was considered by the radiologist (Fig. 9). Gastroscopic examination was impossible because of spasm at the cardia. Cytologic examination showed characteristic lymphomatous cells. The patient was given a course of radiation therapy but died within several weeks because of a pulmonary infarct. At necropsy this was a reticulum cell sarcoma confined to the stomach.

Although the radiologist considered the possibility of lymphoma in this patient,

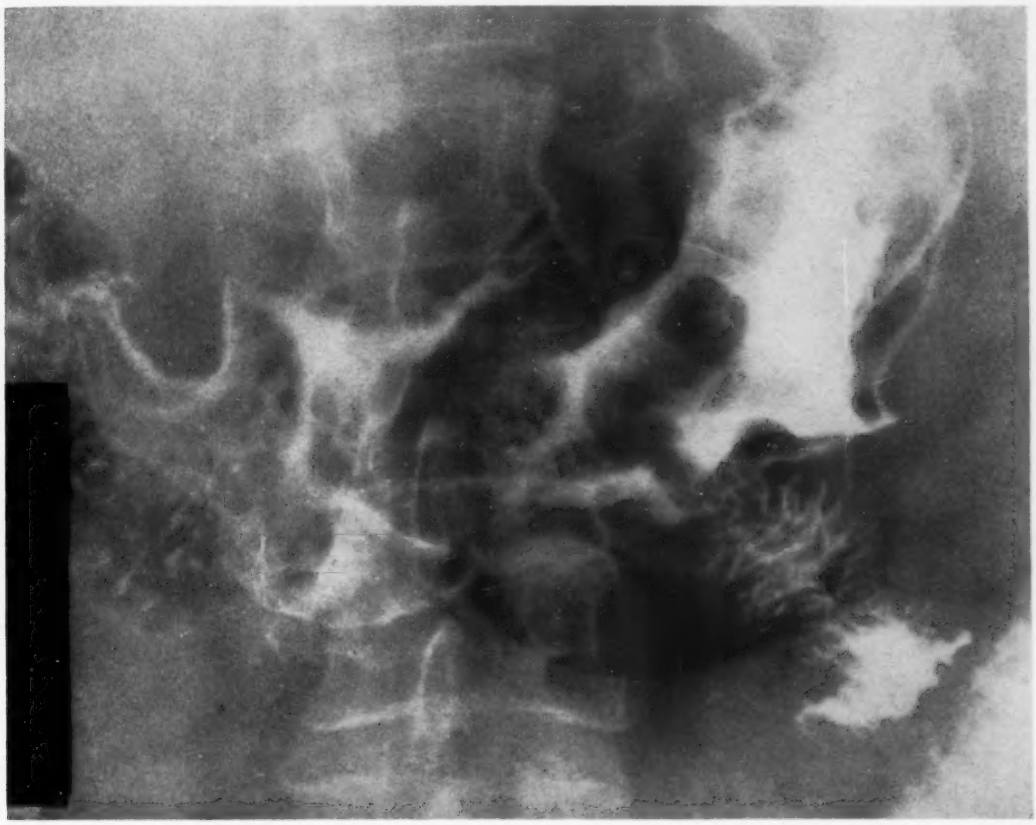

Fig. 9

there is no certain means of diagnosing this disease other than by cytology or biopsy. This is a diagnosis which is indeed worth making; the prognosis is better than that of carcinoma and the therapeutic approach is different.

A thirty-three year old neurotic woman* had recurrent vomiting over a period of three years associated with emotional conflict. Repeated roentgenograms during this period were considered to be negative. Clinically, it seemed very likely that her symptoms were on a functional basis. Unexpectedly, a recent roentgenogram revealed a long pliable irregularity of the distal half of the greater curvature (Fig. 10). It was interpreted as a probable carcinoma. In retrospect, similar findings were present to a lesser extent on the earlier films. Gastroscopy revealed a cobblestone appearance of the distal greater curvature. There was disagreement as to its significance. The differential diagnosis lay between antral gastritis and infiltrative carcinoma. Cytologic examination was repeatedly negative using both chymotrypsin lavage and direct abrasive techniques. In view of the persistent roentgenologic abnormality, laparotomy was recommended. The muscularis mucosae was greatly thickened. Both surgeon and pathologist did not consider this to be a malignant lesion grossly. Histologic examination showed it to be an extensive scirrhous carcinoma, largely submucosal in location, scarcely penetrating to the lumen.

In this rare type of infiltrative malignancy, cells may not reach the lumen. In such instances cytologic diagnosis is impossible. Similar cytologic failures are to be expected in an occasional advanced ulcerated carcinoma which is covered by an impenetrable necrotic membrane.[58] Fortunately roentgenologic examination or gastroscopy enables an accurate diagnosis to be made in many of these cases in which cytology fails.[50]

* Drs. James J. Ahern, Ralph H. Loe and Bryon H. Ward of Seattle cooperated in the study of this patient.

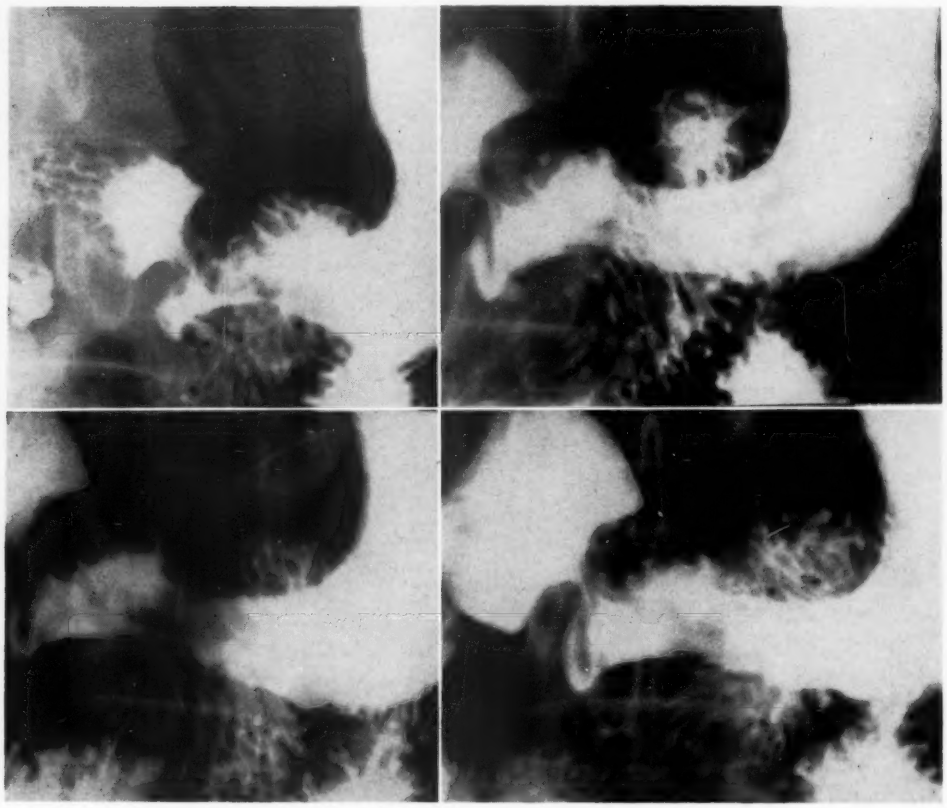

FIG. 10

GASTRIC LYMPHOMA*

Diagnosis of the localized lymphomas of the stomach may be very difficult. The roentgenologic and gastroscopic evidence may be suggestive, but rarely conclusive. On the other hand exfoliative cytology often enables a definitive diagnosis to be made. The criteria for the cellular diagnosis of the lymphoma group are well established.[49] It is important to discover these patients because their disease may be confined to the stomach and may be curable by surgery or radiation.

This fifty-three year old man had a four year history of episodic burning epigastric pain relieved by milk. Roentgenograms of his stomach revealed a large mass in the antrum with a central ulceration; it was considered to be an ulcerated carcinoma (Fig. 11). It had the classical appearance of a diffuse carcinoma by gastroscopy. The cytologic examination established that the malignant cells were of lymphocytic origin. At operation a lymphoma confined to the stomach was found.

The finding of cells arranged individually rather than in clumps with nuclei of lymphoid appearance is characteristic of lymphoma. This criterion enabled a correct preoperative diagnosis in the foregoing patient despite the classical roentgenologic and gastroscopic findings of carcinoma.

This seventy year old woman had lost 30 pounds during a year and had developed a mass in her epigastrium almost the size of a football. On rectal examination a shelf was palpable. A roentgenogram of the stomach showed diffuse nodular neoplastic involvement. Cytologic examination was typical of lymphoma. A laparotomy was therefore performed and the tumor was found to be confined to the stomach. Apparently a calcified uterine fibroid had been misinterpreted as drop metastases causing a rectal shelf. A total gastrectomy was followed

* The cases described in this section have been reported in detail in Reference 49.

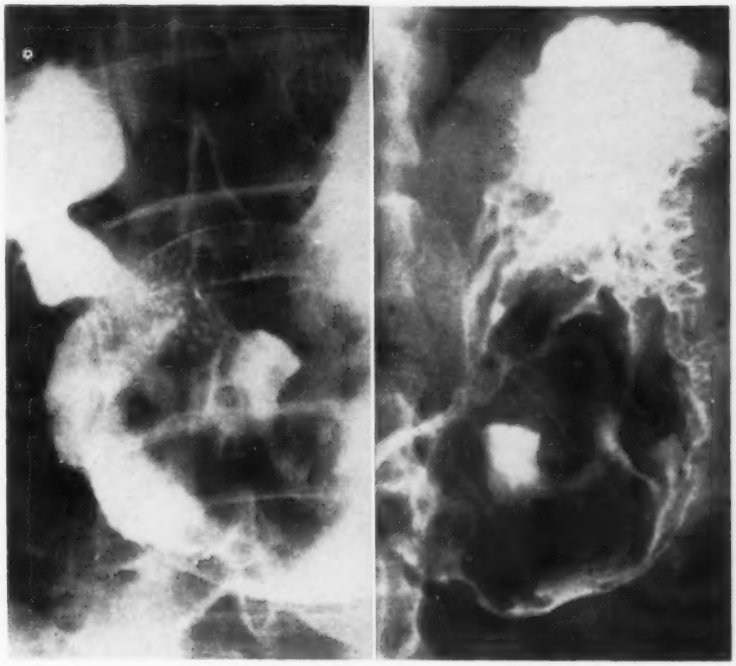

FIG. 11

by radiation therapy. The patient was anorexic after operation and did poorly. Her liver was observed to be enlarging. This suggested hepatic metastases. The patient's appetite improved within a year of operation and the liver size returned to normal, suggesting that the hepatomegaly had been nutritional. A year and a half after operation the patient is able to do her own housework in contrast to her miserable preoperative condition. It was remarked at her recent golden wedding anniversary that she ate a meal as large as that of many of the other guests.

The response of this patient to treatment represents excellent palliation and conceivably a cure. Cytology offered the only certain preoperative diagnosis. No attempt at operation would have been made without this finding.

This thirty-five year old woman had systemic Hodgkin's disease proved by a cervical lymph node biopsy six years previously. For six weeks she was troubled by anorexia and nausea. Gastric roentgen examination revealed a mass on the lesser curvature with a central ulceration. Cytologic examination yielded typical Sternberg-Reed cells establishing the diagnosis of Hodgkin's disease involving the stomach. Sternberg-Reed cells from the cervical biopsy (Fig. 12*A*) were very similar to those found in the smear of gastric cells (Fig. 12*B*). To the author's knowledge, this was the first cytologic diagnosis of gastric Hodgkin's disease. After radiation therapy the lesion regressed completely.

A diagnosis of diffuse Hodgkin's disease with involvement of the stomach was made by exfoliative cytology. Hodgkin's disease may be confined to the stomach and successfully treated by irradiation and surgery. Holmes[14] felt that these tumors occurred almost as often as the rest of the gastric lymphomas and that they were even harder to diagnose roentgenologically. Marshall's figures[26] seem to confirm

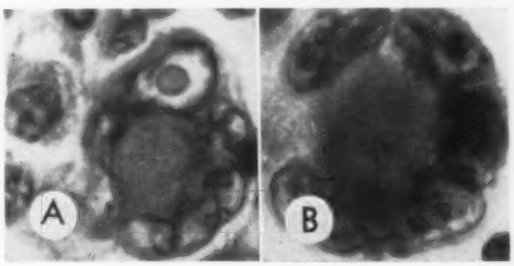

FIG. 12 (*A* and *B*)

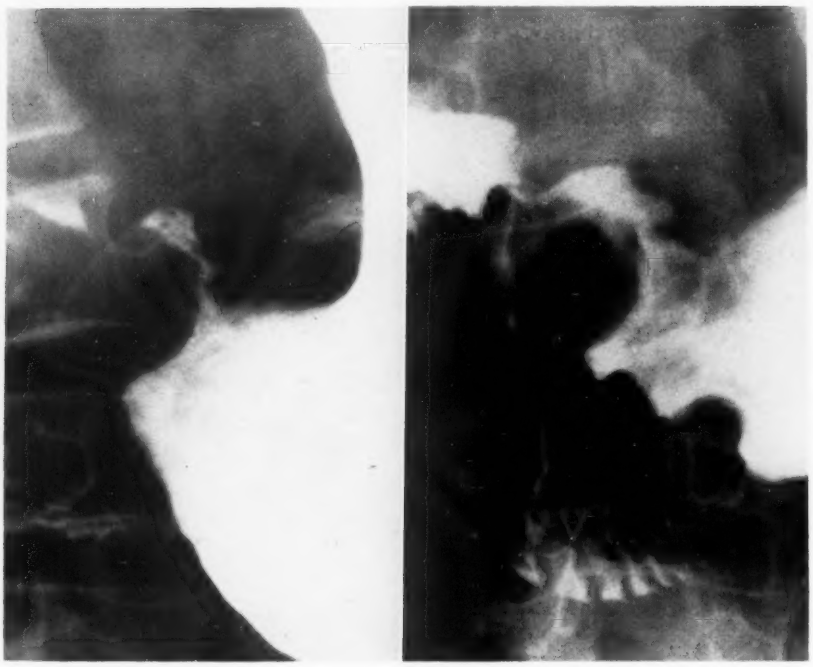

FIG. 13

Holmes' impression of the frequency of gastric Hodgkin's disease. It is to be hoped that others will make the diagnosis of curable, localized Hodgkin's disease of the stomach using these cytologic criteria.

NARROWED ANTRUM

Gastroenterologists, gastroscopists and radiologists alike find it very difficult to determine with certainty the cause of a contracted distal stomach. Cytology may be of particular aid in this area. Several illustrative cases follow.

REPORT OF CASES

This sixty-five year old man* entered the hosptial because of vague gastrointestinal symptoms and increasing weakness. Roentgenologically, the differential diagnosis lay between gastritis and antral carcinoma. Definite malignant cells were found, and at operation the lesion proved to be a carcinoma of the pylorus which had scarcely penetrated the mucosal surface. The pathologist was surprised that cells had been found. A subtotal resection was performed; there were no lymph node metastases. The patient is doing well three years after operation.

The foregoing case had a narrowed antrum of indeterminate etiology which was demonstrated roentgenologically. Exfoliative cytology established the diagnosis and successful surgery ensued. It is of interest that this lesion was relatively inaccessible anatomically. This enabled the comparison of various methods of cell collection. This case was the first which suggested the efficiency of a new technique of cell collection—chymotrypsin lavage.[47]

A sixty-three year old man complained of increasing epigastric pain over the past year. The roentgengram was interpreted as an ulcerating carcinoma of the prepyloric antrum (Fig. 13). The cytology was benign. At operation a benign ulcer was found.

A forty-four year old woman had mild ulcer symptoms of one year's duration. A mass was palpable in the epigastrium. The flouroscopist found an antral ulcer which he considered malignant (Fig. 14). The questionable area was abraded by passing the antral abrasive balloon directly over the lesion under fluoroscopic control. No malignant cells were found. In view

* Reported in detail in Reference 48.

of the roentgen finding of a suspicious antral narrowing with ulceration and the palpable epigastric mass it seemed logical to operate. At laparotomy the gross appearance was that of antral carcinoma. Dissection into the mass enabled frozen sections to be taken which proved its benign nature. It was a benign penetrating ulcer surrounded by a heavy wrapping of swollen omentum. The lesion was successfully resected, and the patient has done well.

The 2 cases described above are examples of a relatively common situation in clinical practice. On the basis of the clinical or roentgenologic evidence, an antral narrowing may be considered malignant only to be proved benign at operation. Cytology is of aid in this situation.

PERNICIOUS ANEMIA

The increased incidence of carcinoma of the stomach in treated pernicious anemia was proved by the classic investigation of Kaplan and Rigler.[20] They reported the value of bi-annual roentgen examination of the stomach in this group of cancer-prone patients.[44] An even greater number of precancerous gastric polyps and early carcinomas will be detected if gastroscopy and cytology are utilized in addition to roentgenologic examination.[45]

REPORT OF CASES

This seventy-one year old man had known pernicious anemia for over five years. He was referred by his private physician for annual cancer screening, despite his asymptomatic status.* On gastroscopy three small polyps were evident. The cytologic findings were of interest. The typical large "bland" columnar cells of gastric origin were obtained. In addition, certain large "active" columnar cells were observed. The latter cells seem more frequent in patients with pernicious anemia who have polyps. On roentgen examination the radiologist could not conscientiously state that polyps were

* This patient was studied through the courtesy of Dr. Jean C. Michel of Seattle.

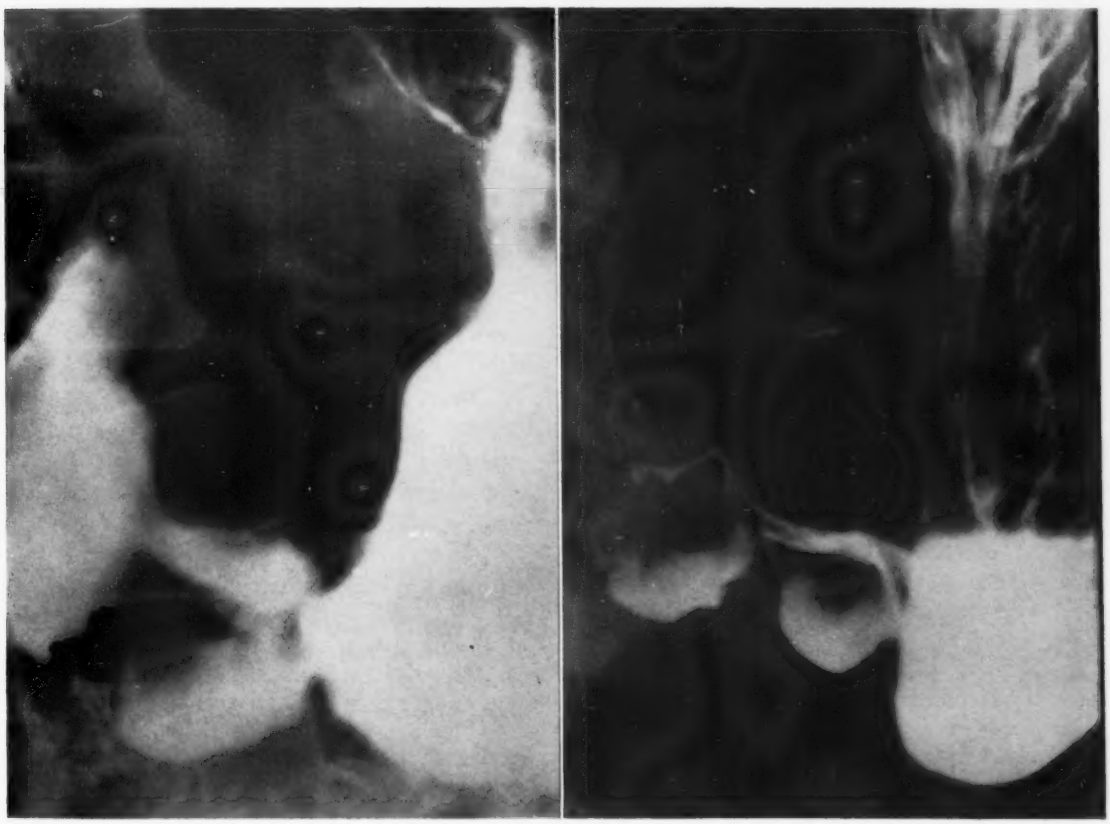

FIG. 14

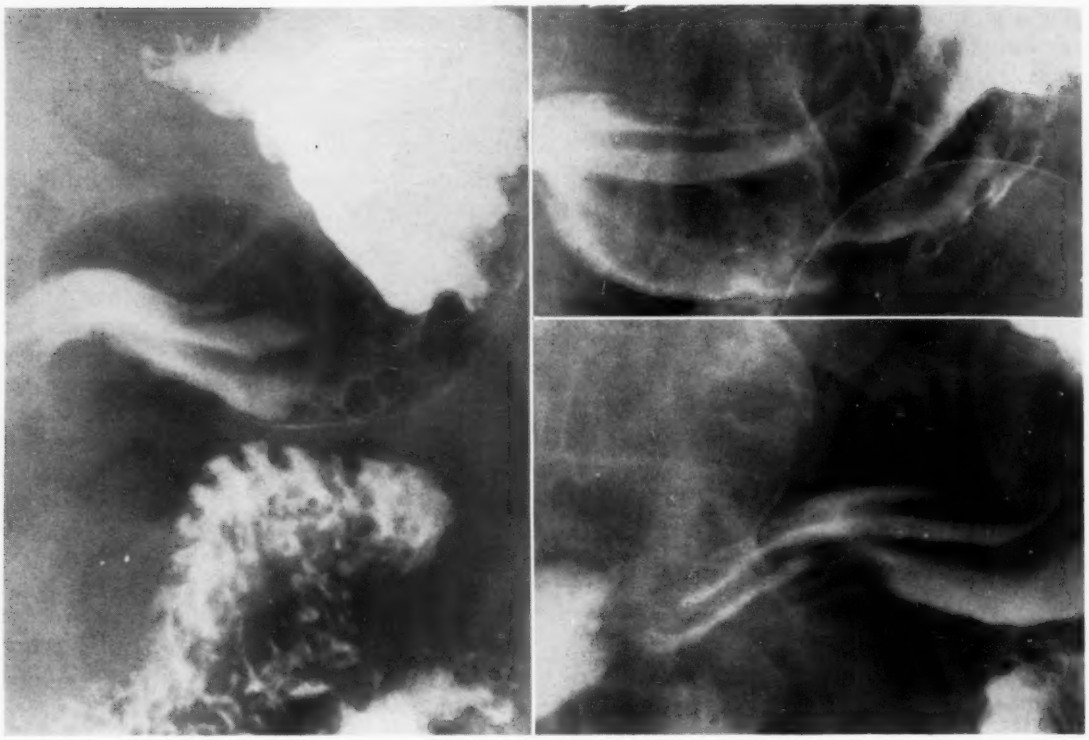

FIG. 15

present and attributed the appearance of the antrum to bubbles (Fig. 15).

In this instance roentgenologic examination showed no gastric polyps, although the cytologic findings suggested their presence. Gastroscopy visualized three polyps.

This twenty-seven year old light-skinned Negro woman* had known pernicious anemia for two years. Her mother had died of gastric carcinoma and it was not known whether she had any blood disease. She was studied extensively at the time of her initial diagnosis in a University Hospital. Roentgen examination was not part of these studies. In the course of an investigation of the value of gastroscopy in treated pernicious anemia, four polyps were discovered. They were benign in appearance and were confirmed by roentgen examination. The cytologic examination showed typical large gastric columnar cells, as well as the active cells referred to above. No malignant cells were seen. A prophylactic operation was performed. A high subtotal gastrectomy yielded an operative specimen containing the four polyps which were grossly benign in appearance. Histologically, they proved to be multicentric intramural carcinomas which had not penetrated to the lumen. Two years have passed since this subtotal gastrectomy. The patient has refused further operation and has successfully carried through a pregnancy. She is apparently well.

* This patient was reported in detail in Reference 45.

CONCLUSIONS AND SUMMARY

Many malignancies of the stomach may be correctly diagnosed by roentgenologic examination. An even higher accuracy is possible if gastroscopy is freely employed.[22] If the pathologist or surgeon cannot definitely determine the malignant nature of some gastric lesions when he has them in his hand, it is not surprising that the radiologist or endoscopist fails on occasion. It is in the problem case that cell study is of greatest value for it is most accurate in differentiating the benign from the malignant.

Gastric exfoliative cytology is useful in differentiating benign from malignant gastric ulcer and in distinguishing heavy folds from infiltrative cancer. It may help estab-

lish the cause of a narrowed antrum. Isolated gastric lymphomas may be definitively diagnosed prior to operation or radiation therapy. The cytologic method also has a definite place in the diagnosis of fundal lesions.

All patients with pernicious anemia should have annual roentgenologic, gastroscopic and exfoliative cytologic studies of their gastrointestinal tract. These studies may uncover premalignant polyps and curable gastric carcinomas.

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GASTRIC ULCER IN THE OLDER AGE GROUP*

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THE subject of gastric ulcer daily confronts every physician, yet the lesion remains one of the most intriguing and controversial in diagnostic and therapeutic medicine. This is partly due to the varied roentgenologic appearances which an ulcer presents and the difficulties with which the roentgenologist is faced in the interpretation as to its malignancy or benignity. Localization of lesions near the pylorus at times proves difficult. The gastroenterologist is confronted with the management of the patient with a potentially malignant lesion. Surgically, the pendulum of treatment of a gastric ulcer has swung from the extremely radical high gastrectomy to a most conservative wedge resection. Diagnostic criteria have been established and, utilizing these, one is able in most cases to determine the exact nature of the lesion; however, errors continue to be made. In this paper these criteria are discussed and one important, but up to now overlooked, factor, the age of the patient, is added. Likewise, the age of the patient is added as a factor in the consideration of therapy.

MATERIAL

The cases included in the analysis were those examined at the Veterans Administration Hospital, Huntington, West Virginia, in a three year span between July 1, 1951, and June 30, 1954. During this period there were 1,968 initial and repeat upper gastrointestinal roentgen examinations performed and a presumptive diagnosis of gastric ulcer was made in 132 patients (6.7 per cent). In this study only the 32 patients who were followed at this hospital were included. Their ulcers were proved either by surgery or by gastroscopic examination. Only 2 of the cases had neither, but the slow definite disappearance of the ulcer roentgenographically, along with the subjective improvement of the patient, was considered adequate positive proof.

The cases of immediate prepyloric ulcers were not included in this study for several reasons. Almost all prepyloric ulcers behave like duodenal ulcers. They occur in younger age groups and localization is difficult. After a few cases of mistaken localization, an arbitrary demarcation line was drawn. Any ulcer located in the distal end of the stomach closer than 1 cm. to the pylorus was considered duodenal. In spite of the arbitrarily drawn 1 cm. line, many ulcers were called gastric, and were found in the duodenum at operation. None was called duodenal and found to be gastric. In reviewing these cases, it was noted that most of these mistaken gastric ulcers were penetrating duodenal ulcers which perforated into the pancreas. The pool of the barium was seen opposite the antrum. Illustrating this difficulty of localization is the following case.

REPORT OF A CASE

CASE 1 (Fig. 2). E.B.L., a sixty year old white male, was admitted December 13, 1953. He had stomach trouble for many years and had tarry stools in 1946. An ulcer was diagnosed in 1948. He did well on medical regimen. Three weeks prior to admission he developed epigastric pain, vomiting and tarry stools. He had lost about 35 pounds in weight in the last six months. Physical examination was essentially negative. Laboratory findings were normal except for a white blood cell count of 14,700. Gastric analysis showed a free acid of 45 and total acid 55. A roentgen diagnosis of deep penetrating ulcer, lesser curvature, distal third of stomach was made. At surgery a penetrating duodenal ulcer was found with a crater 1 cm. in diameter in the pancreas.

AGE DISTRIBUTION

Of the 32 cases studied there were 4 under the age of forty-five. Table 1 shows

* From Veterans Administration Hospital, Huntington, West Virginia.

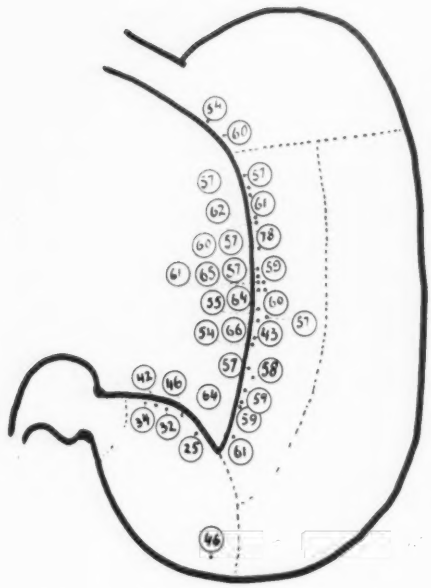


FIG. 1. Schematic drawing showing correlation of age and topography in ulcer cases.

FIG. 2. Duodenal ulcer, incorrectly diagnosed as gastric ulcer.

the age distribution of the 32 cases. The average age was 55.3.

TOPOGRAPHY

In describing the location of the ulcer, the accepted division of the stomach was used with the exception that ulcers which were less than 1 cm. from the pylorus were considered as duodenal ulcers, and the existence of the so-called prepyloric ulcer was discarded as an independent type of ulcer.

There was only one greater curvature ulcer. Of the remaining 31 ulcers there were 6 in the antral portion (distal third), 23 in the middle and 2 in the proximal third. A very interesting conclusion was gained.

TABLE I

AGE DISTRIBUTION

Age	No. of Cases	Per Cent
20–30	1 (25)	3.1
31–40	2 (32–34)	6.2
41–50	5 (45)	15.6
51–60	15	46.8
61–70	8	24.9
71–	1	3.1

All the 6 ulcers found in the antral region were in persons under the age of 50. The average age of the patients having ulcers in the distal third was 38.3, while the average age of those with ulcers in the proximal two-thirds was 60.3 years. This correlation of age and topography is well illustrated in the schematic drawing of Figure 1.

The only case of a greater curvature benign ulcer was found in the antrum. This patient was under the age of fifty. There was one patient aged forty-three in whom roentgenograms showed an ulcer at the midportion of the lesser curvature of the pars media. At operation the resected stomach revealed several small ulcers grouped in the pars media, and two large solitary ulcers in the distal one third.

SYMPTOMATOLOGY

There is a slight difference between the symptomatology of duodenal and gastric ulcers. Duodenal ulcers have a comparatively shorter history with periods of well-being alternating with abdominal distress. In duodenal ulcers pain is the most outstanding complaint. Severe exsanguinating

Table II

Complaints and Symptoms	No. of Cases	Per Cent
Long history of indigestion	26	81.2
Intractable pain	12	37.4
Repeated melena	7	21.8
Exsanguinating hemorrhages	5	15.6
Tenderness	2	6.0
No gastric complaints	6	18.7

hemorrhages, percentagewise, are probably less common. Localized tenderness is very severe. The symptoms in gastric ulcers are almost reversed. All the patients have a history of longstanding vague indigestion sometimes of a number of years'[2] duration. Others had repeated mild hematemeses. Some came under observation because of exsanguinating hemorrhage. It was observed that the gastric ulcer cases, even those with acute hemorrhage, showed very little tenderness. Also it was observed that the tenderness gradually increased in intensity toward the pylorus. In 2 cases, because of the lack of tenderness, the ulcers at the distal end of the stomach were believed to be in the stomach but were found in the duodenum at operation. Table II shows the distribution of the symptomatology of patients studied. During the three years of observation there were a few cases which had to be operated on because of uncontrollable hemorrhage, and in most of those cases a gastric ulcer was found. These were not included in this study because they had no preoperative roentgen studies.

DIAGNOSIS

The perpetual question yet to be answered is the relationship of gastric ulcer to gastric cancer. There are two extreme views concerning this question. One is that no answer is needed, and the differentiation roentgenographically is an unnecessary effort, since by the time an ulcer can be seen, if it is malignant, it is usually far advanced. The other is that every gastric ulcer is potentially malignant; therefore, early operation is necessary.

It may be very difficult, often impossible, to establish an exact differential diagnosis between benign ulcers and ulcerative malignancy. No single criterion is a proof either way. Even the surgeon or pathologist cannot make a definite diagnosis on gross examination, and subsequent histologic examination can reveal the presence of malignant degeneration in the wall of the ulcer.

REPORT OF CASES

Case II (Fig. 3). H.A., a sixty-four year old white male, was admitted with a long history of stomach trouble. A perforated gastric ulcer, which was said to be on the greater curvature, was repaired in February, 1949. In March of the same year roentgenographic examination revealed a normal cardia and media, but a poorly visualized distal one-third. Gastroscopy was negative. Surgery was suggested because of the possibility of cancer, but refused. Patient was asymptomatic until 1951 when he developed mild epigastric pain relieved by amphojel. There was no weight loss, no melena, nausea or vomiting.

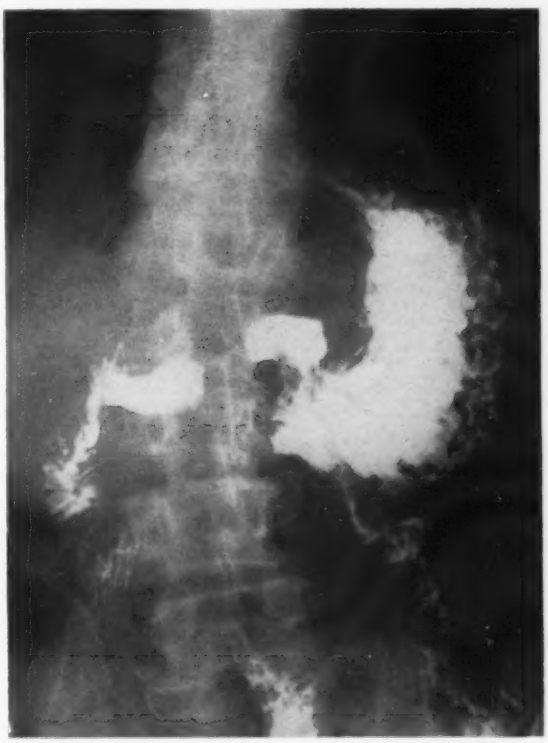


Fig. 3. Adenocarcinoma arising in wall of a gastric ulcer.

Physical examination revealed a thin white male. There was a well-healed abdominal scar. Laboratory findings were all normal. Gastric analysis was reported as 66° free acid and 80° total acid. Roentgenograms showed a large diverticulum on the lesser curvature of the stomach, distal third. The gastric rugae entered into this diverticulum. Interpretation was a benign ulcer.

Surgical exploration in November, 1951, revealed a large mass at the distal end of stomach, lesser curvature side, adherent to the liver, and a subtotal gastrectomy was performed. Gross pathology: benign ulcer 5 cm. from pylorus, 4 cm. in diameter. Microscopic examination: adenocarcinoma arising in the wall of a peptic ulcer. All lymph nodes were negative. Patient did well until July, 1953. He died October 10, 1953. Autopsy revealed generalized carcinomatosis.

This case might offer a strong point in the argument for advocating prompt surgery versus medical trial. Several authors believe it is not enough if the ulcer diminishes in size or even if it disappears under medical treatment. Some feel that if an ulcer disappears under treatment in three or four weeks and the gastric wall shows no residual rigidity, surgery is not warranted. This is shown by the next two cases.

CASE III (Fig. 4, *A* and *B*). 47 972. I.T., a fifty-four year old white male cook, had not had a stomach complaint in his life. On August 2, 1953 he played golf in the morning. Suddenly at 4:30 P.M. he vomited a copious amount of blood, which continued until admission to the hospital at 10:00 P.M.

Physical examination revealed a pale, seriously ill, white male in shock. Blood pressure was 86/76. Pulse rate was almost imperceptible. Complete blood count on admission: red blood cell count, 6,410,000; hemoglobin 21 gm. per cent, and white blood cell count 27,500. The next day the red blood cell count was 5,060,000 and hemoglobin 16 gm. per cent. He was treated medically. The first roentgenogram five days after admission demonstrated a large deep penetrating ulcer at the lesser curvature side of the antrum. A roentgenogram on August 25, 1953, eighteen days after first examination did not demonstrate the ulcer. No recurrence has developed since.

CASE IV (Fig. 5, *A* and *B*). 49 414. H.B., a sixty-two year old white male was admitted on April 8, 1954. He has had many previous admissions because of severe pulmonary emphysema. He had had vague stomach pains and nausea for two weeks' duration but no vomiting, also occasional tarry stools.

Physical examination revealed a poorly nourished, markedly dyspneic, slightly cyanotic male. Blood pressure was 120/70. Examination of the abdomen was negative. Electrocardiogram revealed myocardial damage. The red blood cell count was 3,900,000; hemoglobin was 12.5 gm. per cent; and white blood cell count was 21,400. Other laboratory studies were

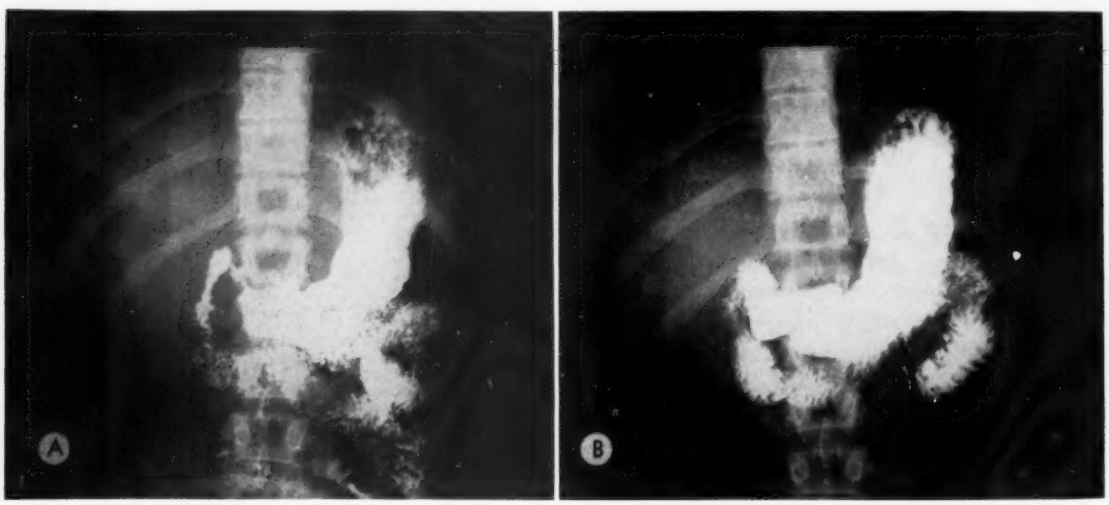


FIG. 4. (*A*) Roentgenogram taken August 7, 1953, showing gastric ulcer in the distal one-third, lesser curvature. (*B*) August 25, 1953, no ulcer demonstrable.

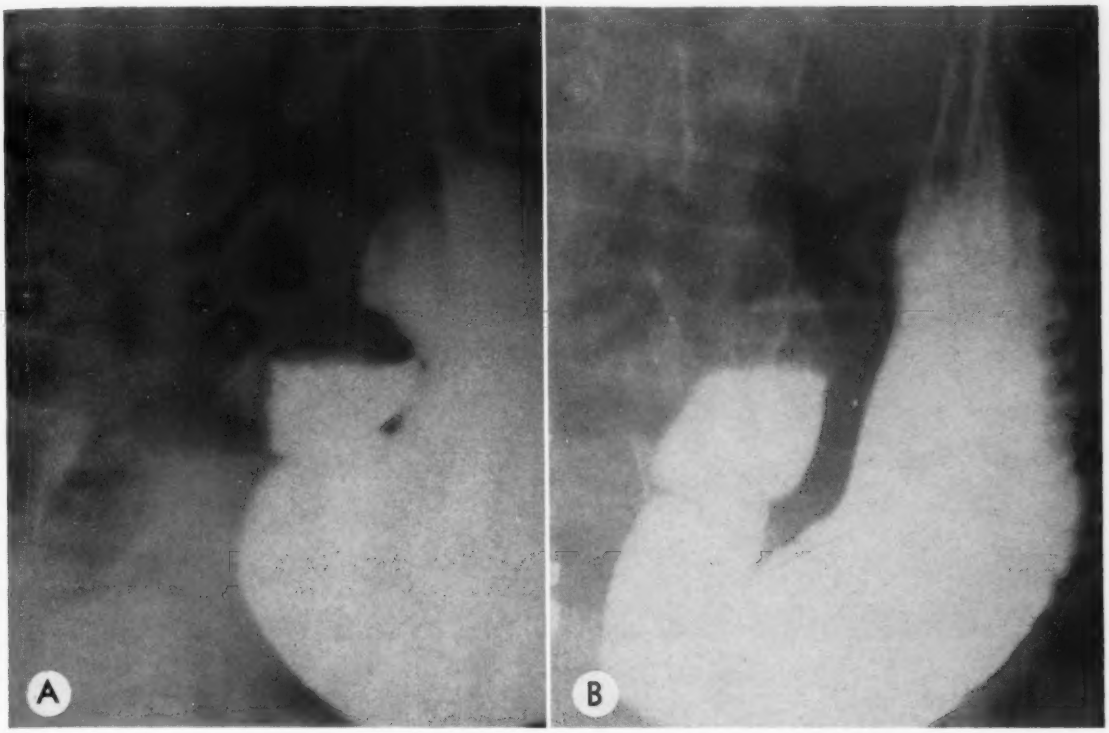


Fig. 5. (*A*) Roentgenogram taken April 30, 1954, showing massive lesser curvature ulcer. (*B*) June 4, 1954, no ulcer demonstrable.

normal. Stool examination for occult blood showed one plus. Gastric analysis revealed free acid of 23°, total acid of 37°. Gastroscopy was not done because of the general condition of the patient. A roentgenogram on April 30, 1954 demonstrated a large ulcer on the lesser curvature side of the stomach, 4 cm. in diameter. On medical regimen all the patient's symptoms abated. On May 18, 1954, a roentgenogram showed the ulcer to be much smaller. On June 4, 1954, the ulcer was no longer visible.

The criteria for differentiation have been established. According to these, the rugal folds in a benign ulcer extend to the margin of the ulcer.

Case v (Fig. 6, *A* and *B*). 49 249. W.K., a fifty-eight year old white male shoemaker,was admitted March 12, 1954. He stated that he had a "cast iron" stomach all his life. On March 10, 1954, he had a massive gastric hemorrhage. Laboratory data on March 12, 1954, showed the red blood cell count to be 2,330,000, and hemoglobin 7.5 gm. per cent. Three days later the patient was still bleeding. At this time his red blood cell count was 1,560,000 and hemoglobin 5.5 gm. per cent. Roentgenograms showed a large lesser curvature ulcer in pars media. Medical regimen was carried out, but because of continued bleeding surgery was performed on April 12, 1954. A lesser curvature ulcer, 1.2 by 1.0 cm. in diameter, at the junction of the lower and middle one-third of the stomach was found. Microscopic diagnosis was benign gastric ulcer.

In active cases, because of the surrounding edema, the folds fade out gradually toward the edge of the ulcer.

Case vi (Fig. 7, *A* and *B*). 48 916. W.R., a sixty year old white male, was admitted on January 23, 1954. He had had stomach trouble for years and was hospitalized for it in 1951. The roentgen findings have been negative since 1949. His complaint at this time was of continuous distress of three to four weeks' duration, not relieved by diet or alkalies. There was a loss of 25 pounds in weight recently.

Physical examination revealed a poorly nourished white male. Examination was otherwise negative. Laboratory studies were normal. Free acidity was 51°. Roentgen examination on

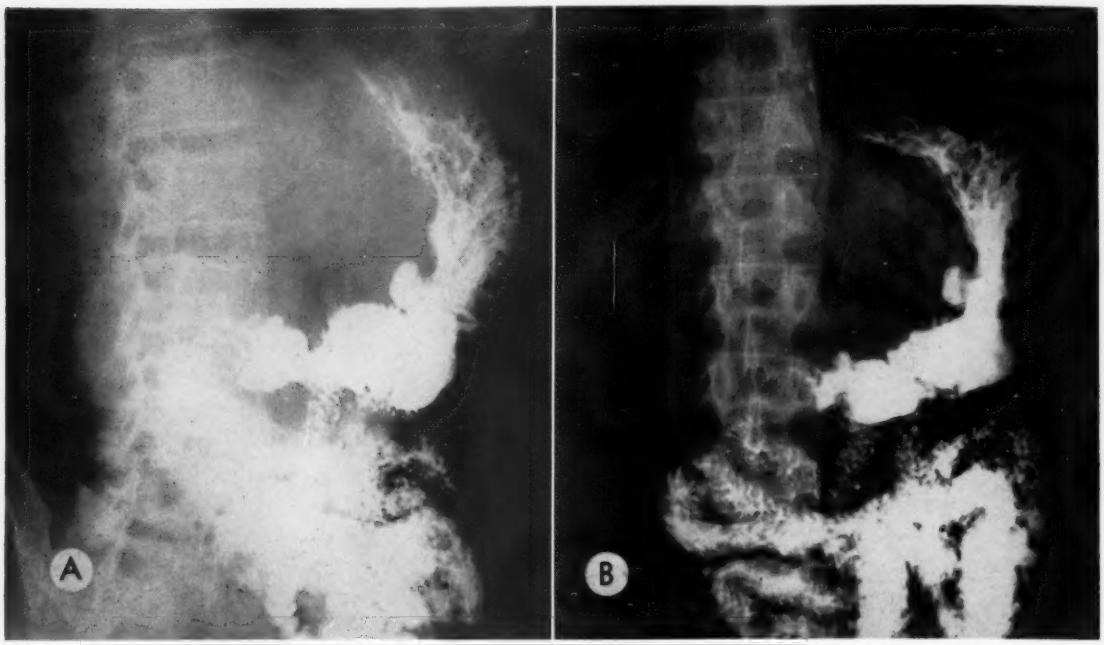


FIG. 6. (*A* and *B*) Benign ulcer showing rugal folds extending to the margin of ulcer.

January 20, 1954 revealed a large high lesser curvature ulcer with a smaller ulcer in the antrum. Both were surrounded by an indurated area. At surgery on January 26, 1954 there were several ulcers on the lesser curvature with indurated margins. All were benign.

In cases of malignancy the rugal folds are supposed to end abruptly a short distance from the margin of the ulcer. There were 2 cases in this series which presented this picture. At operation and on histologic examination, both of these patients were found to have benign ulcers. In Case VIII not only the rugae ended abruptly but a Carman's meniscus sign was seen, which definitely indicates malignancy.

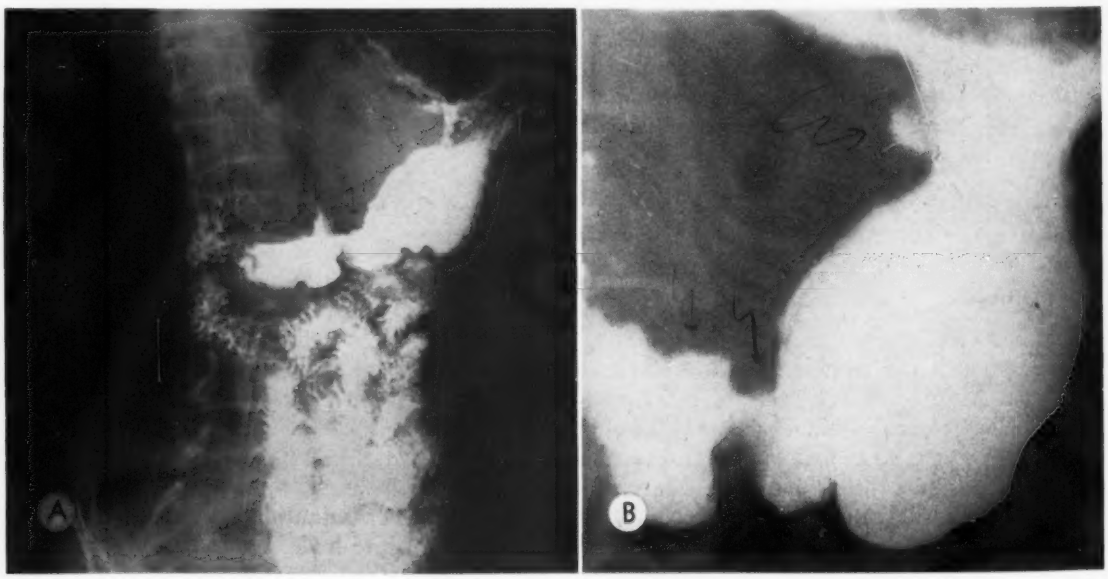


FIG. 7. (*A* and *B*) Multiple benign ulcers with surrounding edema obliterating rugal folds.

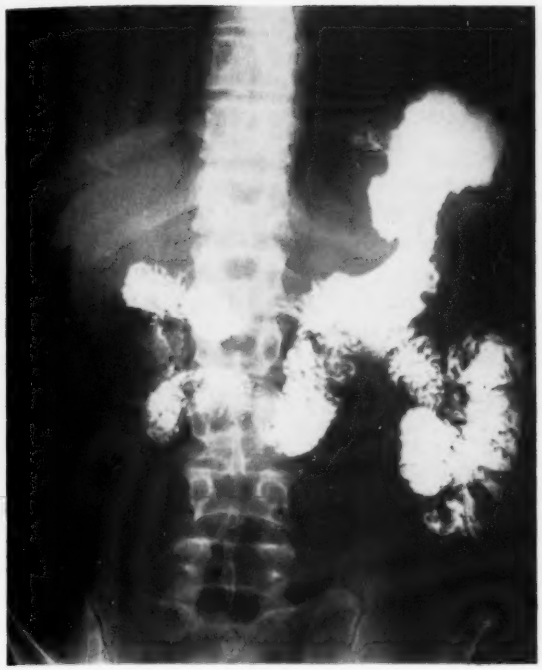


Fig. 8. Massive lesser curvature ulcer with obliteration of rugae considerable distance from edge. Benign ulcer.

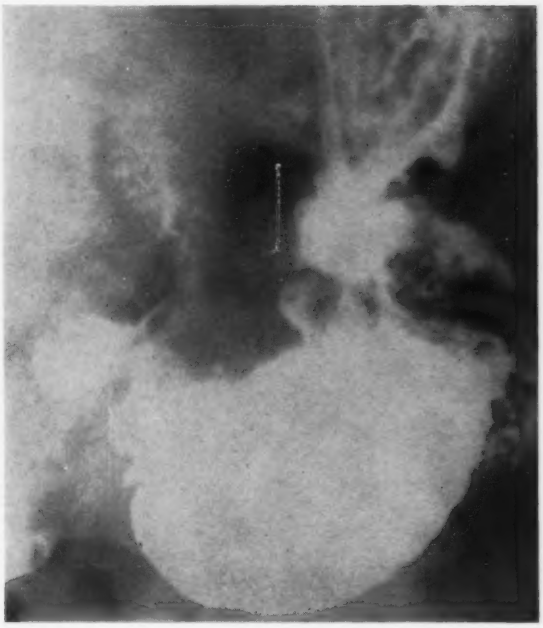


Fig. 9. Multiple ulcers lesser curvature with meniscus sign. Benign ulcers

Case vii (Fig. 8). G.O.B., a fifty-four year old white male, was admitted on May 9, 1952, with a history of vague epigastric distress for two years. Three days prior to admission he vomited a minimal amount of blood. He, also, had had tarry stools on about a dozen occasions in the past year. There was a weight loss of 30 pounds in six months. Physical examination revealed a vague mass in the epigastrium with epigastric tenderness. Laboratory data were normal. Gastric analysis showed 57° total acid and 29° free acid. Roentgen examination showed on the greater curvature side of the stomach a cauliflower-like lesion at the junction of the cardia and pars media. On the lesser curvature side of the stomach just distal to the esophagus, opposite the above lesion, there was a large diverticulum. The roentgen diagnosis was carcinoma. At surgery a large ulcer 2.5 cm. in diameter just below the esophagus on the lesser curvature side was found and a total gastrectomy was carried out. Microscopic diagnosis was benign ulcer.

Case viii (Fig. 9). 47 127. W.F., a sixty-one year old white male, was admitted on October 27, 1952. He first noted epigastric pain of increasing severity in July, 1951. At the time of admission the patient required narcotics for the pain. He vomited everything he ate. He had lost 30 pounds in weight. Physical examination revealed a thin, acutely ill patient. The epigastrium was tender. Laboratory data were normal except for a white blood cell count of 14,550. Gastric analysis revealed a free acid of 6° and a total acid of 21°. A roentgenogram showed at the midportion of the lesser curvature a deep ulcer crater about 1.5 cm. at the base. This ulcer had undermined edges (meniscus sign). Opposite the ulcer the greater curvature was rigid and irregular, suspicious of a filling defect. The lesion was considered malignant. At surgery on November 3, 1952, two ulcers were found. One was close to the pylorus, measuring about 1.0 by 2 cm. in diameter. The other was at the mid-lesser curvature, measuring 2.5 by 2 cm. in diameter. Histologically, both ulcers were benign.

At times the direct and definite signs of the ulcer are misleading. Case ix (Fig. 10) W.P., 45 365 illustrates this. The conversion of rugae is one of the most definite signs of ulceration and is a positive indication of benignity. In Case ix not only was there an ulcer shown roentgenologically, but also seen by the gastroscopist. At op-

FIG. 10. Conversion of rugae at site of ulcer. (No ulcer found at surgery.)

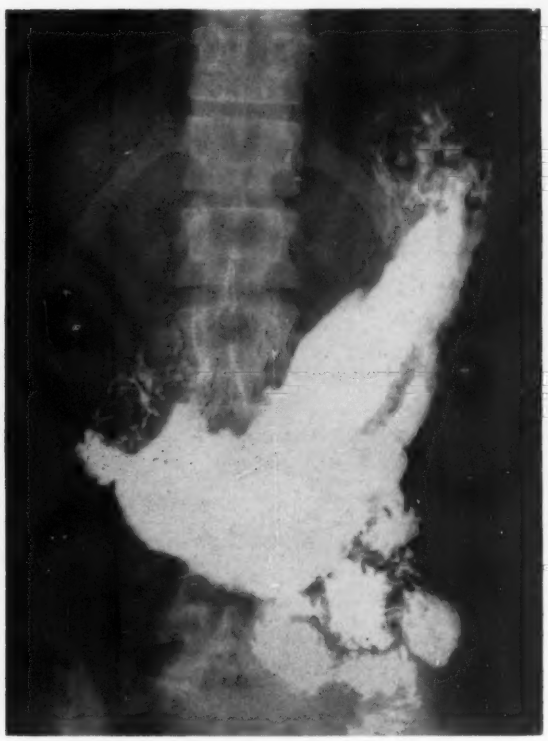


FIG. 11. Multiple ulcers—all benign.

eration five days later no gastric pathology was seen.

Multiplicity almost always indicates benignity. This is seen in the following case.

CASE X (Fig. 11). 49 541. L.C., a forty-three year old male, had a diagnosis of chronic duodenal ulcer made in 1948. During the three to four months prior to admission he had no relief from pain, was nauseated most of the time and vomited frequently. There was a weight loss of 23 pounds. Physical examination revealed only epigastric tenderness. Laboratory data were normal. Gastric analysis revealed free acid of 69° and total acid 86°. A roentgenogram showed numerous ulcers on the lesser curvature. At surgery multiple gastric ulcers, grossly benign, were found. There were 5 ulcer craters, one large and one in the mid-lesser curvature with other ulcers surrounding it about 2–3 cm. distance. All ulcers were benign microscopically.

In addition to the above discussed direct

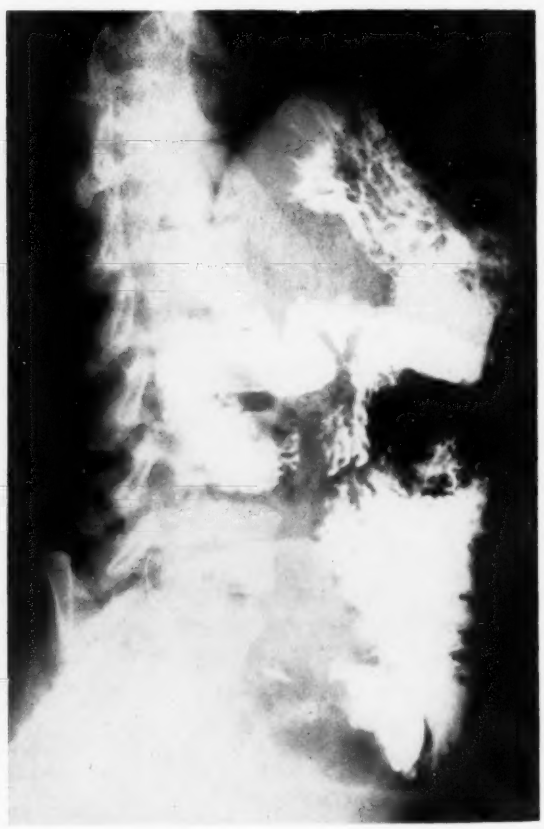


FIG. 12. Rigid filling plus, greater curvature opposite lesser curvature ulcer. Benign.

signs, there are a few indirect or secondary signs which, however, often are not reliable. In one case a large, rigid, somewhat undermined area was seen on the lesser curvature side. Opposite this was a cone-shaped rigid filling plus. This case was considered to be a malignancy with ulceration, especially because the patient was rather young. At surgery a benign lesser curvature ulcer 1.5 cm. in diameter and 2 cm. above the pylorus was found (Case XI, Fig. 12).

It is well known that the gastric peristalsis is sluggish or the stomach even atonic in gastric malignancy, while there is normal or hyperactive peristalsis in benign ulcerations. It was found that in the older age group when the ulcer is farther from the pylorus, the peristalsis tends to be sluggish, sometimes even absent. The incisura on the opposite curvature pointing to the ulcer was seen in very few cases. In Case XI there were two such incisurae opposite an ulcer which was shallow and indurated. The two incisurae formed a cone-shaped filling plus between them which suggested that the ulcer was on the greater curvature side. In the 32 cases no hourglass deformity was seen. Similar deformity is the gastrospasm with foreshortening of the stomach, which gives an appearance of an annular obstruction, especially since there is absence of peristalsis in the stomach above the lesion. There were 4 cases of this deformity and in all 4 cases the mistaken diagnosis of malignant lesion was made. Two are presented.

CASE XII (Fig. 13). 49 587. W.C.A., a sixty-four year old male, was admitted on May 6, 1954 with a six month history of upper abdominal pain. There was no hematemesis, nausea or vomiting, and no weight loss. Physical examination revealed mild epigastric tenderness. Laboratory data were normal. Gastric analysis showed 28° of free acid and a total acid of 92°. Roentgen diagnosis was annular malignant infiltration with a pencil-mark lumen. At surgery a mid-lesser curvature ulcer, funnel-shaped, measuring 1.1 by 1.2 cm. was found. Histologically it was a benign gastric ulcer. The rigidity and the channel-like lumen were produced by spasm and surrounding edema.

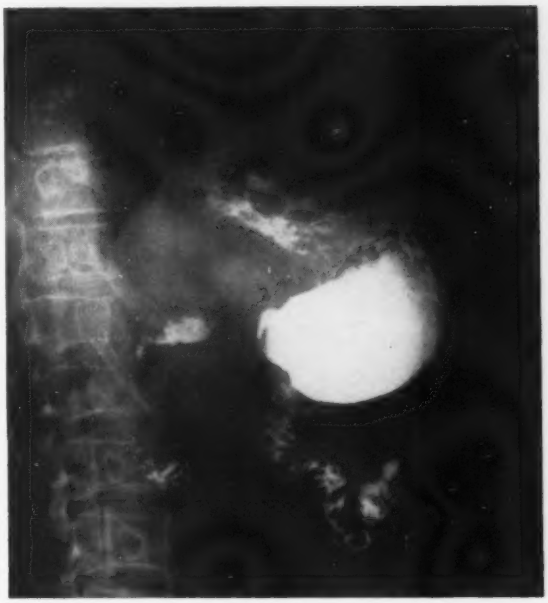


FIG. 13. Spasm and edema in distal stomach, producing pencil lumen. Benign ulcer.

In Case XIII not only the roentgen findings but the gastroscopic findings were those of malignancy.

CASE XIII (Fig. 14). 49 772. J.B., a sixty-six year old colored male, was admitted on March

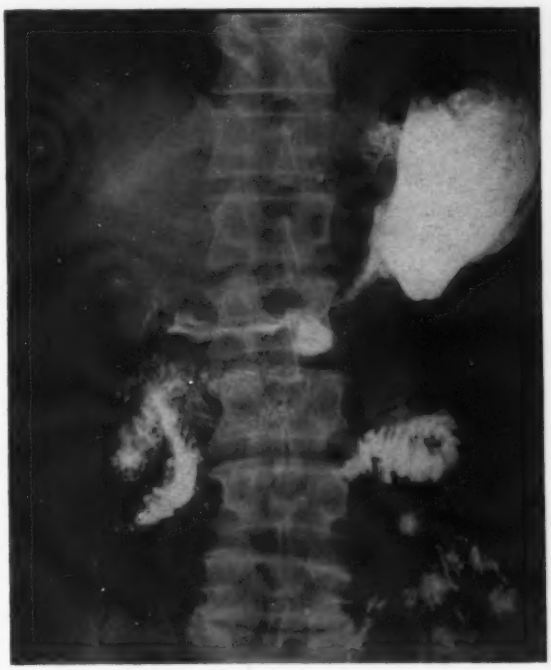


FIG. 14 Multiple ulcers with spasm of distal stomach. Benign ulcers.

17, 1954 for headaches, dizziness, and weakness. He had no definite gastrointestinal complaints. Physical examination revealed hypertension and prostatic enlargement. Laboratory data were normal. Gastric analysis showed free acid of 60° and total acid of 80°. A roentgenogram on March 23, 1954 showed an annular constriction with probable ulceration involving the distal half of the stomach. On gastroscopy the gastroscope would not pass beyond the pars media because of obstruction. At surgery on April 13, 1954 a mid-lesser curvature ulcer, 1.2 cm. in diameter, was found. In addition, there were two ulcers in the antrum, slightly smaller in size. Microscopically all were benign.

Large gastric ulcers near the cardia more often simulate malignancy than those nearer to the antrum. At first all these cases were operated on and giant benign ulcers were found with marked induration of the wall. Interestingly, many of these patients had very few symptoms other than mild indigestion for many years. The farther the ulcer is from the pylorus, often the fewer symptoms they produce.

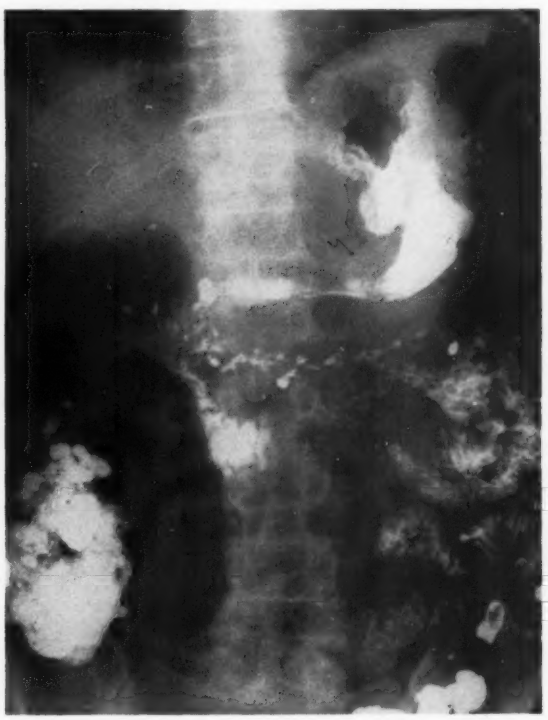


FIG. 15. Massive ulcer, proximal one-third of stomach, with edema and greater curvature spasm simulating carcinoma. Benign ulcer.

CASE XIV (Fig. 15). J.D., a fifty-seven year old white male, was admitted on May 11, 1953 complaining of indigestion of two years' duration. He had tarry stools frequently. Physical examination was negative except for pallor and tachycardia. Patient had a red blood cell count of 1,720,000; hemoglobin 5 gm. per cent and a white blood cell count of 19,300. Gastric analysis showed 24° free acid and 45° total acid. From May 11, 1953 to May 20, 1953, 4,000 cc. of blood was administered. A roentgenogram on May 20, 1953 showed a large ulceration of the stomach on the lesser curvature just beneath the esophagus. There was a filling defect on the greater curvature opposite the ulcer, considered to be malignant. On gastroscopy, May 22, 1953, an ulcerating necrotic carcinoma was seen. At surgery a large ulcer on the lesser curvature 3 cm. in diameter, surrounded by an inflammatory mass about 5 cm. in diameter, was found. The base of the ulcer was in the pancreas. Microscopically it was a benign ulcer.

CASE XV (Fig. 16). 48 123. S.W., a sixty year old white male, was admitted on October 1, 1952. He had had stomach trouble since 1935. At this time he complained of mild epigastric pain of six months' duration, tarry stools, and nausea and vomiting of six weeks' duration

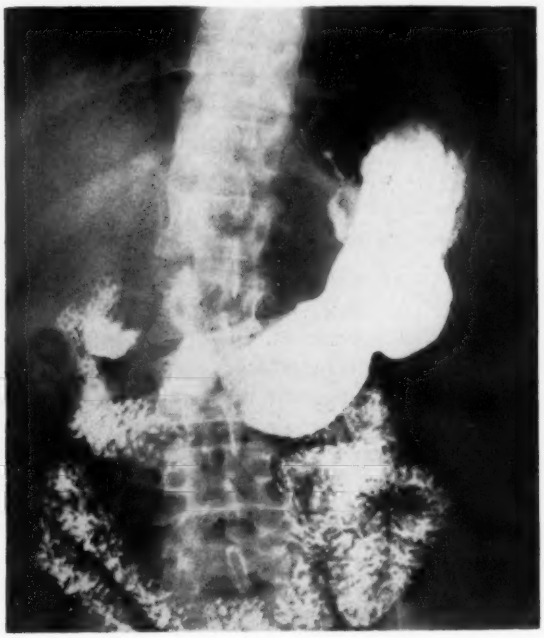


FIG. 16. Benign ulcer with inflammatory mass, proximal lesser curvature of stomach.

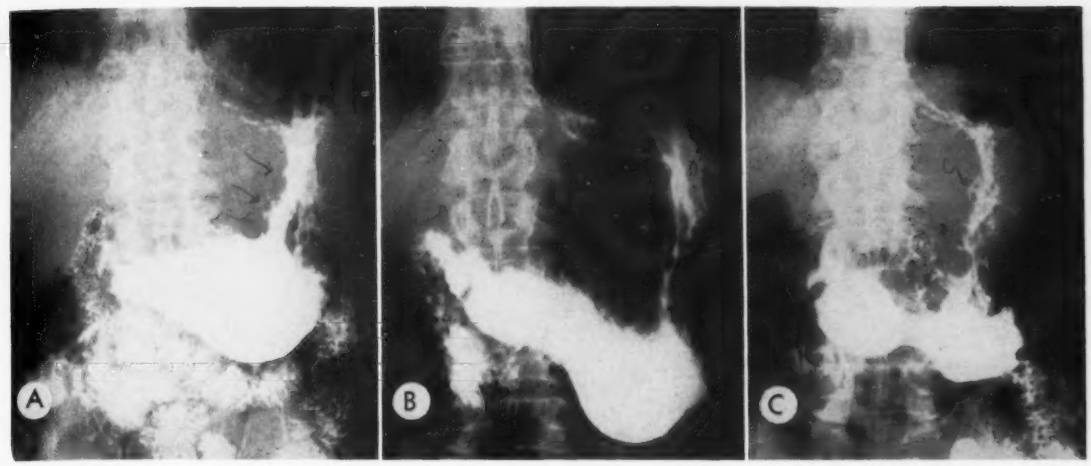


Fig. 17. (*A*) Lesser curvature ulcers, May, 1953. (*B*) Healed ulcer, October, 1953. (*C*) Recurrence of ulcer, August, 1954.

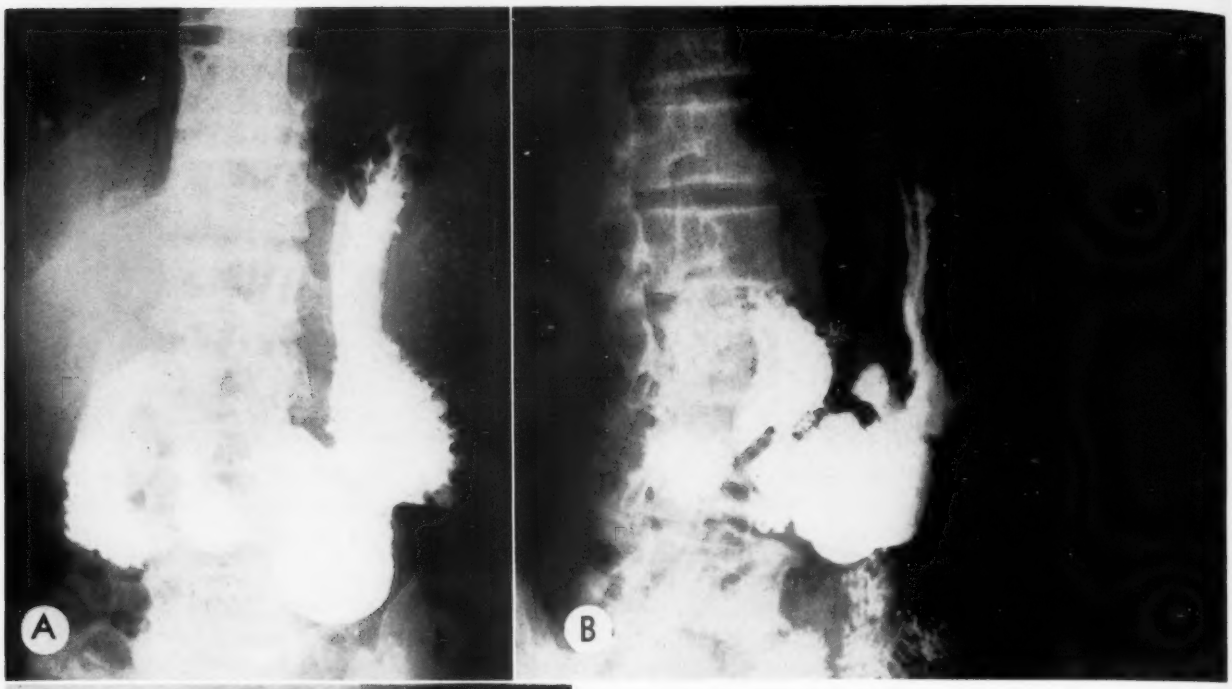
with moderate hematemesis and a weight loss of 30 pounds. Physical examination revealed an acutely ill patient, malnourished, edentulous, with severe asthma and marked epigastric tenderness. Laboratory data showed a red blood cell count of 3,960,000; hemoglobin was 10.5 gm. per cent. Gastric analysis revealed a free acid of 0° and a total acid of 14°. A roentgenogram demonstrated an ulcer crater with indurated margins near the esophagus on the lesser curvature. After five days on medical ulcer regimen the red blood cell count was 2,980,000 and hemoglobin 9.5 gm. per cent. At surgery a lesser curvature ulcer 1.5 cm. in diameter, almost at the esophagus, was found. It was surrounded by an inflammatory mass. The area of induration with the ulcer was 6 cm. in diameter. A benign ulcer was the microscopic diagnosis.

It is known that gastric ulcers have a tendency to recur. One case has been followed for several years. The primary complaint of this individual was hematuria (Case xvi. C.R. 50 283. Fig. 17, *A*, *B* and *C*). On initial admission on August 29, 1954, he complained of gastrointestinal disturbance and a roentgenogram showed a gastric ulcer. Since then he has had but minimal or no stomach disturbance. On every admission for his urinary pathology he has had gastrointestinal series for academic interest only. The recurrence and disappearance of the ulcer was seen four times.

Case xvii (Fig. 18, *A*, *B* and *C*). 49 559. S.S., a fifty-seven year old white male, was admitted on May 1, 1954 because of acute onset of epigastric pain. He had a sixteen year history of gastric discomfort which was relieved by food and diet. He had a diagnosis of gastric ulcer made in 1938 and had severe symptoms two to three years ago. He also had tarry stools one year ago. In May, 1953, a roentgenogram showed a small, apparently healing gastric ulcer. He had an acute onset of pain and vomiting two weeks prior to admission. Physical examination was negative. Laboratory data were negative. Gastric analysis showed free acid of 14° and total acid 24°. Roentgenograms in May, 1954, revealed a large penetrating ulcer at the mid-lesser curvature. An air pocket was seen at the top of the ulcer in the upright position. This patient was operated on because of the marked increase in the size of the ulcer and because the air-fluid-barium layers in the ulcer in the upright position were mistaken for a sealed off perforation.

COMMENT

The purpose of this study is to present for consideration the following factors in the interpretation of gastric ulcers: (1) *The age factor:* The average age of the studied cases was 55.3 years. Gastric ulcer in the older age group tends to be benign. Only 1 in 32 cases was malignant. Because of this it is felt that immediate surgery in the older age group is not necessarily indicated. It is felt that gastric ulcers in the



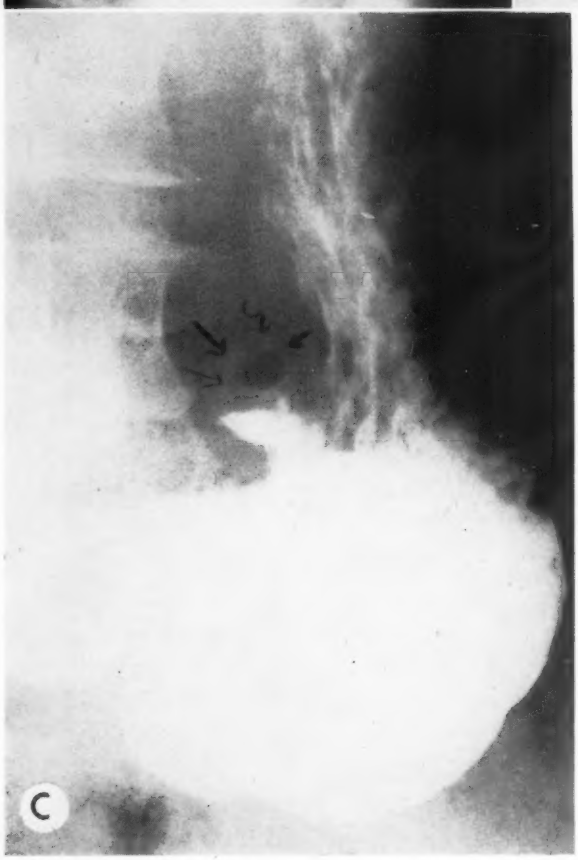


FIG. 18. (*A*) Small ulcer, lesser curvature seen May 22, 1953. (*B*) Massive ulcer one year later (May 3, 1954). (*C*) Air bubble in summit of ulcer, same date.

older age group deserve a trial of medical management before surgery is decided upon. (2) *Gastroscopy:* It is only a diagnostic adjunct and no more than that, even in the hands of an expert. In 6 cases gastroscopy was negative and surgery showed the large ulcers that were seen by the radiologist. There were several cases where gastroscopy suggested malignancy and the surgical finding proved benignity. The gastroscopist should confirm the presence or absence of a lesion and not interpret it histologically. (3) *Location:* The discrepancy between the roentgen and surgical findings in regards to the location of the ulcer is rather great. This could be explained by the curling of the lesser curvature caused by induration or spasm, or both. The ulcers at surgery are often closer to the pylorus than seen on the roentgenograms. (4) *Symptomatology:* In gastric ulcers the symptoms are not characteristic. Many ulcers produce very few symptoms and often date back ten to twenty years. Many of the cases were admitted for completely different reasons and the ulcers were found incidentally as a result of gastrointestinal examination performed as a part of a complete check-up. Hemorrhage, either melena or exsanguinating, is a common complication.

Characteristic roentgenograms of cases illustrating the criteria of benignity and malignancy are presented with the fallibility of these. Thirty-two cases of proved gastric ulcers of older age group are discussed.

SUMMARY

1. In the older age group, gastric ulcers tend to be benign.

2. Often, mild symptoms exist for years before a diagnosis of ulcer is made.

3. Gastric ulcers tend to heal quickly, but are apt to recur. A healed gastric ulcer leaves little, if any, roentgenologic deformity, as compared with the scarring and deformity of a duodenal ulcer.

4. Dogmatic roentgenologic diagnoses as regards malignancy or benignity are unwarranted.

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HEMANGIOMA OF THE STOMACH WITH A ROENTGENOLOGIC DIAGNOSTIC POINT

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HEMANGIOMAS of the stomach are quite rare, only 25 having been reported to date. The present report includes apparently the first case in which the diagnosis was made preoperatively, a roentgenologic sign to be looked for in all cases of obscure gastrointestinal hemorrhage, and a review of the literature.

REPORT OF A CASE

Mr. G.E.H., a thirty-one year old white male student, during the course of a routine physical examination, was discovered to have a hemoglobin of 50 per cent. He never had any gastrointestinal complaints and denied melena. Two gastrointestinal series were negative. The presence of melena was then reported and confirmed. A third gastrointestinal series revealed the presence of a lesser curvature mass in the center of which were what appeared to be phleboliths. Past history and systemic review were essentially negative. Physical examination was nonrevealing, and no abdominal masses were palpable.

On admission to the hospital hemoglobin was 8 gm., red blood cell count 3.2 million, white blood cell count 7,100 with a normal differential. Feces were repeatedly positive for occult blood. The total protein was 7.3 gm. per cent, albumin 4.53 and globulin 2.77 gm. per cent. Serology was negative. On the basis of the melena, the intragastric mass, and phleboliths in the mass, shown roentgenologically, a preoperative diagnosis of hemangioma was made by the surgeon (Fig. 1).

Two thousand cubic centimeters of whole blood transfusions were administered prior to operation. At exploration a mass, the size of a walnut, was palpable in the pyloric region of the stomach on the anterior wall; it was irregular, soft, and freely movable. A gastrostomy was performed, and the presence of a hemangioma confirmed. The lesion was excised in toto, and routine closure performed with pyloroplasty. The convalescence was uneventful, and he was discharged on the eighth postoperative day.

Microscopic analysis of the specimen revealed an angioma with a superficial ulceration and four calculi. The calculi consisted of calcium phosphate and calcium carbonate.

COMMENT

Benign tumors of the stomach are infrequent, and hemangiomas form a small part of the entire group. The incidence was statistically computed by Eliason and Wright[5] in an investigation of over 1,000 benign tumors of the stomach. Myomas comprised 57.3 per cent and papillomas 7.8 per cent; "polyps," adenomas, cysts, and lipomas each formed 5 per cent; the frequency of angiomas was 1.7 per cent.

The age incidence of reported cases has varied from twenty to sixty-seven years, with a slightly higher incidence in the fifth and sixth decades. The first case reported was by Lammers[13] in 1893.

The formation of a hemangioma of the stomach is ascribed to embryonic rests of mesodermal tissue, and may be of a capillary or cavernous variety. The tumor is located in the submucosal region, and is typically bluish-black or reddish in appearance; on palpation the mass is smooth, soft, round, and has been well described as a "mass of worms." In a few instances a pedicle has been present, and 2 cases have

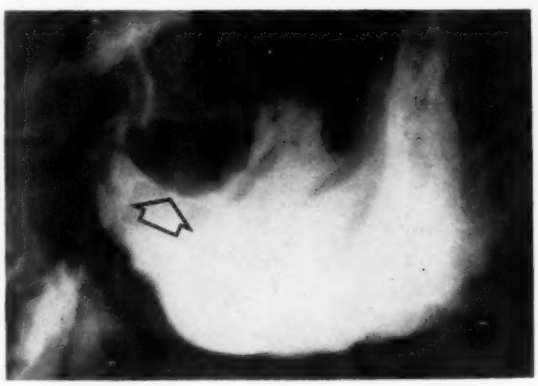

FIG. 1. Hemangioma of the pyloric antrum with phleboliths.

been reported in which a sarcomatous change was present. The lesion has been found in all portions of the stomach, and may be multiple.

Symptomatology will vary depending on the size and location of the hemangioma, and may easily mimic an acute or chronic gastritis, pylorospasm, carcinoma, or more commonly a bleeding gastric or duodenal ulcer. An acute phase with hematemesis, melena and epigastric pain is usually described, with a period of recovery to be followed by a recrudescence of all complaints. Eusterman and Senty[6] in 1922 reported 4 cases, in one of which a tumor 7 by 5 cm. produced signs of intermittent pyloric obstruction.

In the cases of Sherrill and Graves,[19] and Freeman,[7] a mass was palpable in the epigastrium. The latter reported a hemangioma 20 cm. in diameter that arose from the lesser curvature and almost entirely replaced the gastrohepatic omentum. Chevallier and co-workers'[3] case contained four separate lesions, as evidenced roengenographically; the diagnosis was made at autopsy.

Therapy will vary depending on the size and location of the angioma, and may range from simple excision to gastric resection. Surgical excision, especially following episodes of hematemesis and melena, has been curative in all cases. In only one instance, that of Morton and Burger,[18] was the lesion considered too widespread in the stomach for any type of excision.

SUMMARY

1. The twenty-sixth case report, and the first in which the diagnosis was made preoperatively, of hemangioma of the stomach is presented.

2. The presence of phleboliths within a gastric mass, confirming the diagnosis, is a roentgeologic sign not previously described.

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GALLSTONES IMPACTED IN THE STOMACH AND DUODENAL BULB DEMONSTRATED BY ROENTGENOLOGIC EXAMINATION

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THERE has been a sufficient number of reports of lower small intestinal obstructions due to gallstones and the incidence of occurrence is high enough so that all radiologists can recall cases of their own and will consider the possibility of gallstones in any such obstruction without apparent cause in an elderly patient. Although medical literature contains frequent oblique references to gallstones in the stomach, when a study is made only a few can be located. No reports are found where gallstones are demonstrated roentgenologically in the stomach. Described below are two such cases.

REPORT OF CASES

CASE I. F.P., a fifty-seven year old male, was admitted to St. Clare's Hospital on May 6, 1954, with a history of upper abdominal discomfort of five days' duration and a one day history of nausea, eructations and vomiting "brownish material." An appendectomy had been performed fourteen years previously. Tenderness was elicited in the epigastric area on physical examination. Laboratory study revealed 5,100,000 red blood cells, hemoglobin 16.7 gm., 14,800 white blood cells with normal differential, icteric index 7.6, and a blood sugar of 247 mg. per hundred cubic centimeters. Also, there was a 4 plus glycosuria and a trace of acetone in the urine. Upper intestinal tract roentgen examination revealed in the gastric antrum a 4 cm. oval defect which was smooth in outline but with irregular barium-filled crevices on its surface (Fig. 1 and 2). The adjacent rugal pattern was normal. The mass was fixed in position and could not be displaced with deep palpation. A bezoar was considered briefly, but the impression was that this represented a polyp.

Operation was performed on May 11, 1954. The entire right upper quadrant was found to contain a mass of adhesions with a cholecystogastric fistula extending to the pylorus. The fistula was dissected and upon exploration the filling defect proved to be a large gallstone in the antrum completely obstructing the stomach (Fig. 3). The procedure elected was a subtotal gastric resection with a Pölya anterior gastrojejunostomy, and cholecystectomy following gastrotomy and removal of the stone. The resected specimen had an irregular laceration near the pylorus, proximal to which the mucosa presented granularity suggestive of chronic inflammation.

The patient felt exceptionally well during his postoperative period and was discharged after seventeen days.

CASE II. (Submitted by Dr. James T. Case of Santa Barbara, California.) J.S., a sixty-three year old male, was admitted to Battle Creek Sanatorium with complaints suggestive of cholecystitis. Upper intestinal tract roentgenologic series revealed a large smooth defect in the gastric antrum which changed in position during examination (Fig. 4). The diagnosis was that of a polypoid tumor.

Operation was performed in October, 1924. A mass of adhesions was found surrounding the gallbladder, involving the duodenum, liver, transverse colon and omentum. The gallbladder appeared to be full of stones. A gastroenterostomy was done for immediate relief and a large gallstone found in the stomach was removed. The stomach was examined carefully and no evidence of cholecystogastric fistula was found. Laboratory analysis showed the calculus to be definitely a gallstone.

Recovery apparently was uneventful, but no additional data are available.

A majority of gallstones responsible for mechanical intestinal obstruction enters the duodenum through fistula formations and obstructs the lower ileum.[2]

Occasionally fistula formation to the

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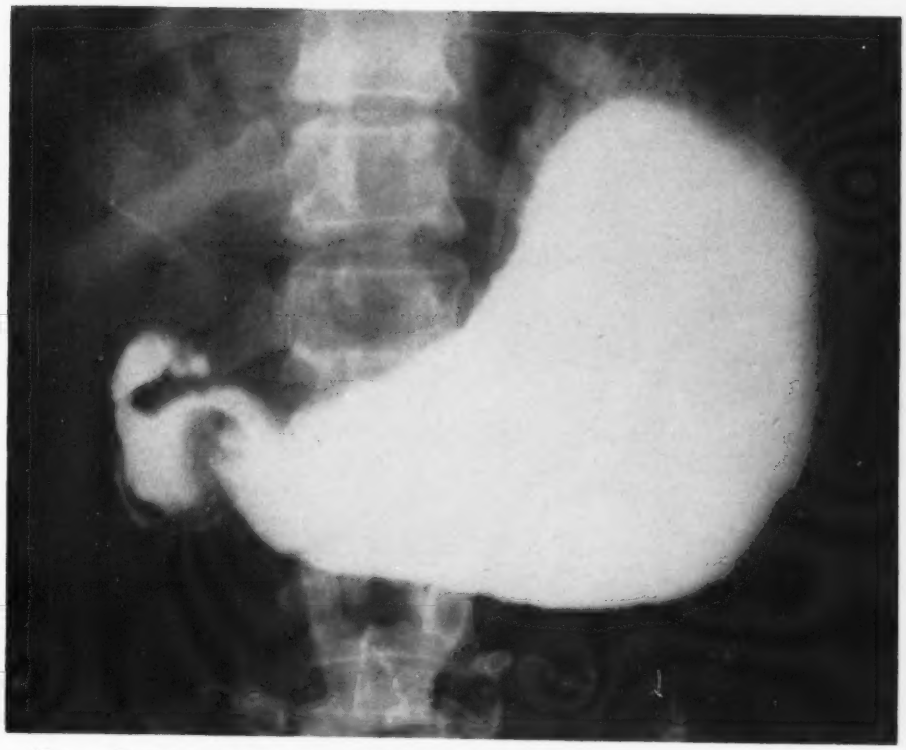

FIG. 1. Case 1. Outline of the gallstone which had entered the stomach through a cholecystogastric fistula.

FIG. 2. Case 1. Pressure spot roentgenogram of the gallstone impacted in the gastric antrum.

FIG. 3. Case I. Calculus removed from the stomach.

stomach may occur with the presence of diffuse inflammatory and adhesive processes which protect against peritonitis during perforation. This was the situation in the first patient. Because of the pathologic report, there is no doubt that the calculus had been in the stomach much longer than would seem apparent by the brief history. In the second patient presence of a cholecystoduodenal fistula may be assumed to have been the mode of entry into the intestinal tract with subsequent passage to the stomach. Although a stone may appear too large to pass through the pylorus, surgeons may at operation force a large duodenal stone into the stomach without difficulty.

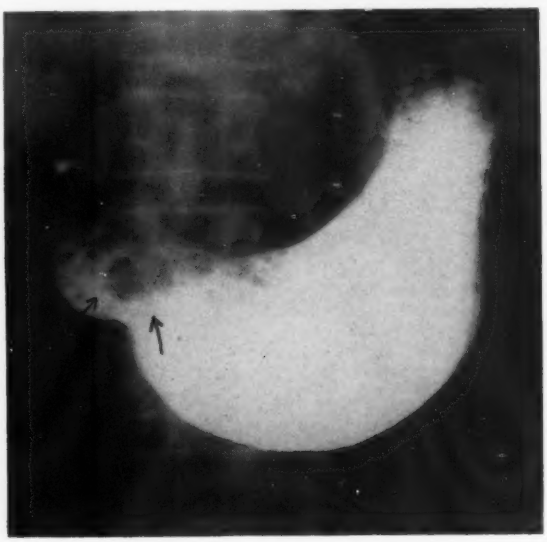

FIG. 4. Case II. Outline of the gallstone which had entered the stomach by way of the duodenum.

Wakefield, Vickers and Walters,[19] Roth and co-workers, and Courvoisier and Naunyn (quoted by Gråberger[8]) report the occurrence of 20 gastric fistulae in their separate collections of 603 cholecystoenteric fistulae, an incidence of 3.1 per cent. Isolated reports of cholecystogastric fistulae have been made by Martin,[11] Smith,[17] Rankin,[13] and Pohlandt,[12] the last two diagnosed by air and barium in the biliary tract.

The first recorded case of a gallstone found ulcerating into the stomach was that of Beaussier in 1770.[1] Since that time the number of reports has remained meager. One is described where a large gallstone was noted to form the base of a pyloric ulcer.[5] An internal biliary fistula between the gallbladder and the pyloric portion of the stomach and a 1.5 cm. calculus located in a small cystic cavity of the stomach wall separated from the lumen were found by Reiman and Bloom in a patient who had died with pneumonia and empyema.[14] In Hertz'[10] collection 3 cases of large stones impacted in the pylorus are mentioned and 1 is listed by Thompson.[18] In DeMole's patient, an obstructing calculus in the duodenal bulb was demonstrated roentgenologically, while a second smaller stone, not so demonstrated was found in the stomach at operation.[4] There had been formation only of a biliary-duodenal fistula which indicates retrograde passage into the stomach. No published reports of gallstones in the stomach demonstrated roentgenologically, however, have been found.

Apparently only 31 cholecystogastric fistulae and 8 gallstones found in the stomach have been recorded and most of these occurred more than thirty years ago. With the increase in elective gallbladder surgery, the incidence of occurrence should drop

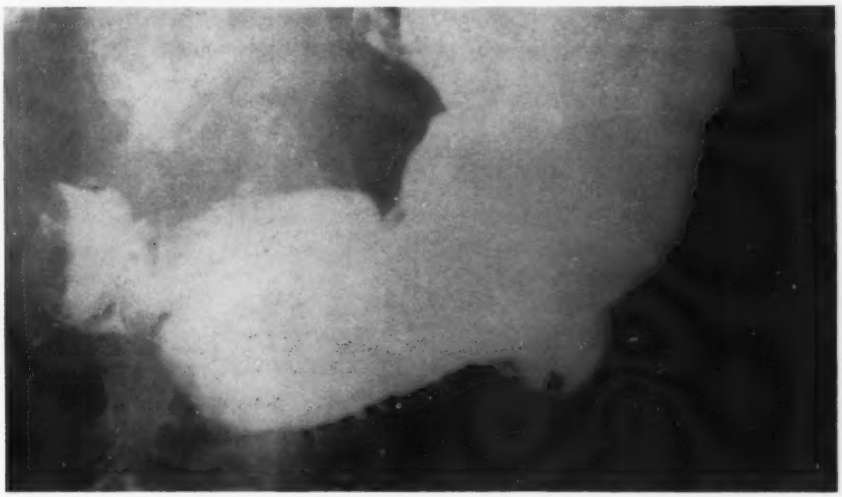

Fig. 5. Case III. A pressure defect on the duodenal bulb can be seen. This was produced by the gallstone shortly before penetration.

even lower. Accordingly, in the differential diagnosis of gastric intraluminal defects, gallstone may be omitted without any significant risk of error.

Approximately 50 per cent of fistulae between the gallbladder and intestinal tract occur in the duodenum.[2] Despite this high incidence, there are only a few cases in the literature of findings of stones in the duodenal bulb. Only 6 such cases have been proved roentgenologically.[6,15] Therefore, it is felt that the following case of gallstone

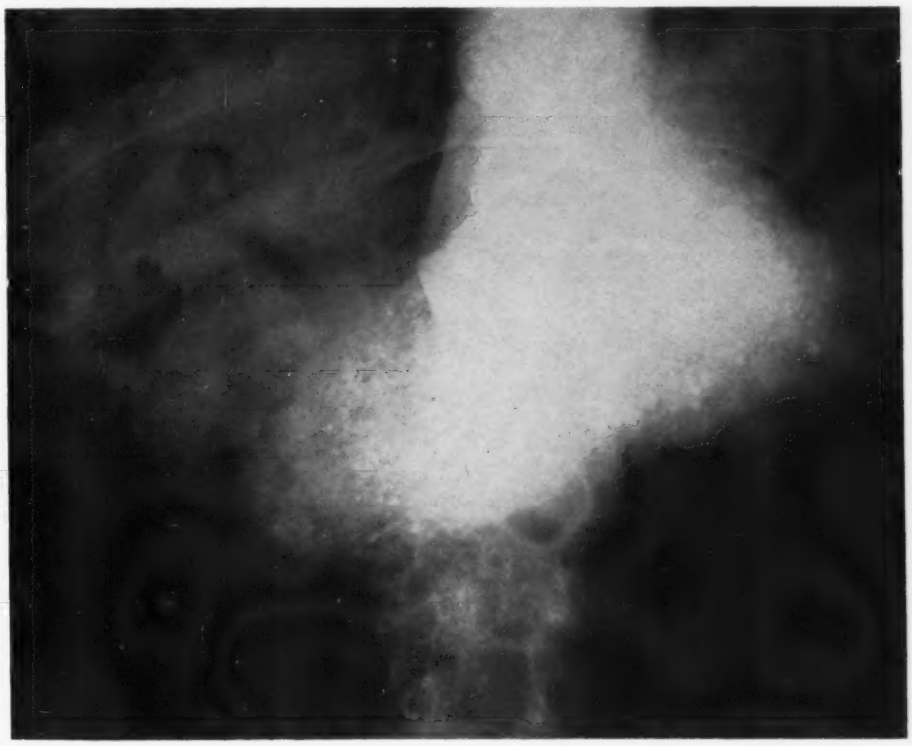

Fig. 6. Case III. Air in the biliary tract and telepaque retained in a distended stomach.

impacted in the duodenal bulb merits publication.

CASE III. P.S., a seventy-four old woman, was admitted to St. Clare's Hospital in May, 1954, for removal of a sebaceous cyst of her back. Her physician palpated a questionable mass in the right upper quadrant and requested a film of her abdomen which revealed only elevation of the right diaphragm leaf and enlargement of the liver. Her white blood cell count was 12,700 with 48 lymphocytes per 100 cells. A gastrointestinal series done elsewhere on September 30, 1954, revealed a pressure defect on the duodenum (Fig. 5).

She was re-admitted on October 15, 1954, with complaints of anorexia and epigastric pain for one week. Pain had begun as a dull ache occurring usually after meals, accompanied by sour eructations. Just prior to admission, it increased in severity and was followed by vomiting green material. She admitted similar episodes of pain and vomiting five years before. On physical examination there was enlargement of the liver and epigastric tenderness. Blood chemistry values were normal. Gallbladder series revealed nonvisualization of the viscus, air outlining the biliary tract and complete retention of the contrast medium in a dilated stomach (Fig. 6). Following gastric lavage, an upper intestinal tract study revealed a dilated stomach and complete duodenal obstruction in three hours and a 3 cm. density, partially calcified, in the right lower quadrant. A 24-hour film showed barium filling the bilary tract, a calculus in the common duct and a very large defect outlined in the duodenal bulb area (Fig. 7).

FIG. 7. Case III. Barium outlining the gallstone in the duodenal bulb, the biliary tract and a calculus in the common duct 24 hours after ingestion of the meal. The density in the right lower quadrant is a large calcified appendiceal fecalith. A broken lumbar puncture needle behind L_4 had been present for twenty-five years.

At operation in October 20, 1954, a large inflammatory mass was found in the right upper quadrant with a cholecystoduodenal fistula. A 7 by 4 by 4 cm. gallstone was found distal to the pylorus obstructing the duodenum (Fig. 8). A gastrotomy was performed and the calculus forced into the stomach and removed. The fistula was not dissected. A 2.5 cm. calculus was noted in the appendix which was removed. The remainder of the intestine was explored and no other calculi were found. Laboratory analysis of the appendiceal calculus proved this to be a fecalith.

Fourteen days postoperatively the patient experienced eructations, nausea, vomiting, abdominal distention and foul yellow stool. A roentgenogram of the abdomen showed obstruction in the proximal ileum. Exploration revealed the obstruction to be due to a Richter's hernia of the umbilicus which was reduced and repaired. No calculi were found.

Following the second operation, recovery was uneventful and the patient was discharged on November 20, 1954.

As additional stones may exist either above or below the initial obstructing stone,[7,9] this possibility was suggested by the density in the right lower quadrant, although its configuration was not characteristic. Second stones may escape even careful examination of the intestinal tract during surgery, and therefore suspicion was aroused again when postoperative obstruction occurred. In both instances, however, this was not the situation.

Fig. 8. Case III. Cut-section of the calculus removed from the duodenal bulb.

SUMMARY

1. Fistulae to the stomach occur in 3 per cent of all cholecystoenteric fistulae. Thirty-one have been recorded.

2. Medical literature contains reports of only 8 gallstones found in the stomach at operation or autopsy and none which were demonstrated roentgenologically. These enter usually through a cholecystogastric fistula. Because of elective surgery, this diagnosis should become even more rare.

3. Two cases are presented of gallstones impacted in the stomach which were demonstrated by roentgenologic examination.

4. A case is presented of a very large gallstone impacted in the duodenal bulb demonstrated roentgenologically before and after perforation.

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THE RELATIONSHIP OF STENOSIS TO ROKITANSKY ASCHOFF SINUSES OF THE GALLBLADDER*

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ROENTGENOGRAPHIC visualization of Rokitansky-Aschoff sinuses was first reported by March in 1948.[13] In May of 1948, a similar case was seen in the Department of Radiology at The Buffalo General Hospital; this case was reported by one of us (G.J.C.[3]) in 1950. Since that time we have collected 4 additional cases, 3 of which have been proved histologically. We feel that the presence of stenosis of the proximal portion of the gallbladder is a most important factor for the cholecystographic demonstration of Rokitansky-Aschoff sinuses.

The detailed histologic picture has been described by Boyd[4] in his inimitable style. His description of the normal gallbladder mucosa is as follows: "When the fresh gallbladder, immersed in water, is observed by reflected light, the picture is a remarkable and beautiful one. Tall graceful folds and membranes, gossamer-like in their delicacy, can be seen floating in the fluid-like seaweeds in a marine pool. The entire inner surface is divided by these membranes into a series of polygonal spaces, each of which resembles a little courtyard, surrounded by high though delicate walls." He goes on to state: "In disease, the picture may undergo a complete change, and the delicate membranes become thickened, debased, and may finally disappear. The gallbladder has no submucosa. In the normal organ, no glandular structures pass from the mucosa into the underlying tissue, but in chronically inflamed gallbladders outpouchings of the mucosa may often be seen to pass for a varying distance into the muscularis and sometimes even into the perimuscular layer."

These hernia-like outpouchings were first described by von Rokitansky[18] in 1842. Aschoff demonstrated the muscular layer of the gallbladder to be sparse and irregular in structure. Vessels penetrate the muscular layer through its interstices which are often wide and allow the muscoa to herniate through. Halpert showed the morphologic changes to be acquired; he found no such changes in serial sections of 10 fetal gallbladders. Robertson and Ferguson[14] in an analysis of 495 gallbladders concluded that the main factors in the formation of crypts (diverticula) are advancing age plus the peculiarly loose and complex distribution of muscle bundles in the gallbladder wall, the isolated physiologic response of these fascicles to normal stimuli, and the increased pressure produced by dysfunction or mechanical conditions. They found that over 50 per cent of the gallbladders in patients over thirty years of age (including non-diseased gallbladders) had histologic evidence of Rokitansky-Aschoff sinuses. No reference to stenosis, septa or other abnormalities was made.

We have reviewed all of the available reported cases in the radiologic literature. There are 14 cases reported by others; W. D. Buchanan has been kind enough to allow us to see his material of a case prior to publication. In addition we have 5 cases to add (1 has been reported in 1950). All cases present a typical cholecystographic appearance, *i.e.*, relatively good function with a concentric thin irregular ring of contrast medium about the lumen of the gallbladder. In all but 4 of the published cases and Buchanan's case, a definite area of stenosis or constriction was present in the proximal portion of the gallbladder. The Rokitansky-Aschoff sinuses were distal to the stenosis. As noted in a previous publication we feel that the area of stenosis or constriction may be important in the development of very large sinuses which can be

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visualized roentgenographically and represents a very important aid in the diagnosis of Rokitansky-Aschoff sinuses.

March's case was a white female, aged twenty-seven. Oral cholecystography demonstrated good function with typical findings as described above including a segment of constriction in the proximal gallbladder.

Ackerman[1] reported a case of a white female whose gallbladder revealed very large Rokitansky-Aschoff sinuses, but no evidence of stenosis. No histologic proof was obtained.

Åkerlund and Rudhe[2] reported the cholecystographic findings of a fifty-two year old white female. The examination was carried out three times with typical findings including an area of stenosis near the ampulla of the gallbladder. He noted better filling of the marginal or concentric ring of contrast medium following the fat meal. The best demonstration of Rokitansky-Aschoff sinuses was obtained after subcutaneous administration of morphine sulphate following fatty meal. Histologic proof was not obtained.

CHART I

CASES REPORTED BY OTHERS

	Author	Date Published	Presence of Stenosis
1.	March[13]	February, 1948	yes
2.	Ackerman[1]	August, 1948	no
3.	Åkerlund and Rudhe[2]	February, 1950	yes
4.	Glücker[7]	February, 1951	no
5.	Zinober[19]	January, 1952	yes
6.	Lawler[12]	April, 1952	no
7.	King[9]	———, 1952	yes
8.	King[10]	September, 1952	no
9.	Sukman and Daron[17]	May, 1953	yes
10.	Buchanan[5]	(Personal communication)	no
11.	LaPointe[11]	June, 1953	yes
12.	Kamdar[8]	January, 1955	yes
13.	Robinson and Peters[15]	February, 1955	yes
14.	Ross, Finby and Evans[16]	March, 1955 (2 cases)	yes

CHART II

AUTHORS' CASES

	Date Examined	Presence of Stenosis
1.	May, 1948	yes
2.	March, 1951	yes
3.	October, 1953	yes
4.	November, 1953	yes
5.	January, 1954	yes

Total Number of Cases—20.
Cases with Stenosis—15.

Zinober[19] published the case of a forty-eight year old male with typical cholecystographic findings including a definite stenotic ring in the proximal portion of the gallbladder. There was good concentration of the contrast substance.

Sukman and Daron[17] reported the case of a twenty-two year old Negro male. The cholecystographic findings included good function, a constriction near the ampulla of the gallbladder with Rokitansky-Aschoff sinuses distal to this segment.

Buchanan's[5] case was an adult female. The cholecystographic picture of marked Rokitansky-Aschoff sinuses was present. The case was proved histologically.

Additional single cases of macroscopic Rokitansky-Aschoff sinus formation have been reported by LaPointe,[11] Kamdar,[8] Robinson and Peters,[15] Glücker,[7] Lawler,[12] and King.[9,10] Ross, Finby and Evans[16] have reported two additional cases.

REPORT OF CASES

CASE 1.*A fifty-six year old white female was referred for cholecystography, May 1, 1948. She had a previous admission in 1944 for resection of the left ovary (serous cystadenoma) and total hysterectomy for multiple uterine fibroids. During the next four years the patient had periodic attacks of right upper quadrant pain, nausea and emesis. There was no history of jaundice, dark urine, or light colored stools. These attacks gradually increased in frequency and severity.

Physical Examination: Temperature, 99° F.; pulse, 88; respiration, 20; blood pressure,

* Reported previously.[3]

Fig. 1. Case 1. The shadow of the gallbladder is seen with a constriction just below the neck.

Fig. 2. Case 1. After a fat meal, there is complete emptying of the small upper pouch. Note the thin rim of contrast medium in the wall of the gallbladder.

100/50. The patient did not appear acutely ill. Abdominal examination revealed a well healed scar in the lower abdomen; there was slight generalized distention, but no tenderness. A walnut sized mass was palpated below the right costal margin.

Laboratory Findings: Urinalysis was normal. Erythrocyte count, 4,400,000; hemoglobin, 12.5 gm.; white blood cell count, 12,000; differential count normal. Glucose and urea normal.

Roentgen Findings: Oral cholecystography revealed a gallbladder shadow of slightly less than normal density. There was a constriction just below the gallbladder neck which was persistent both before and after the fat meal (Fig. 1). Following the fat meal the gallbladder shadow contracted with complete emptying of the portion above the stricture while below the stricture there was some extravasation of contrast medium into the gallbladder wall forming a thin concentric ring of contrast substance around the lumen (Fig. 2).

Operation: June 5, 1948. Cholecystectomy revealed a thick walled gallbladder which was removed by subserosal dissection.

Surgical Pathology: The resected gallbladder measured 10 by 3 cm. The wall of the gallbladder was markedly edematous and thickened, measuring 0.5 cm. in thickness. At the fundus there were numerous match-head sized sinuses. The neck portion was narrowed and there were also pea-sized diverticula in this area which were filled with pigmented stones. In the lumen there were about six small cinder-like stones. A microscopic section through the gallbladder wall showed marked chronic fibrous cholecystitis. There were numerous Rokitansky-Aschoff sinuses, some of which were macroscopic in size and contained pigmented concrements (Fig. 3.)

Case II. A thirty-nine year old white female had complained of chronic indigestion and fatty intolerance for many years. Her fatty intolerance had increased during the three months prior to admission on March 3, 1951. The patient had daily nausea and vomiting with right upper quadrant pain radiating through to the back and left scapula. There was no history of fever or jaundice; the stools were normal. The patient had an appendectomy in 1934, with an uneventful recovery.

Physical Examination: A moderately obese female in no acute distress. Temperature, 99.2° F.; pulse, 92; respiration, 18; blood pressure 100/60. Palpation of the abdomen revealed

Fig. 3. Case 1. Low power (×60) photomicrograph of microscopic section of gallbladder wall at the fundus, showing Rokitansky-Aschoff sinuses penetrating the muscular layer and extending toward the outer layers. A, mucosa; B, muscular layer; C, Rokitansky-Aschoff sinuses.

minimal tenderness in the right upper quadrant with some guarding. No masses were palpable.

Laboratory Findings: Urinalysis was within normal limits. Erythrocyte count, 4,500,000; hemoglobin, 13.2 gm.; white blood cell count, 7,700.

Roentgen Findings: Oral cholecystography on March 6, 1951 revealed good function. The gallbladder was divided into almost two separate compartments by a stricture through its midportion. The visualized lower portion demonstrated a radiolucent rim about its circumference, with a very fine curvilinear line of contrast medium just outside of the gallbladder lumen. There was a very distinct, partially calcified calculus within the fundus (Fig. 4).

Operation: Cholecystectomy was performed without difficulty on March 7, 1951. The gallbladder was involved in a thickened process, and midway between the fundus and ampulla was a sharply defined fibrous constriction. The patient made an uneventful postoperative recovery and was discharged March 16, 1951.

Surgical Pathology: The gallbladder measured 9.5 cm. in length, 5 cm. in circumference at the fundus, and 2 cm. in circumference at the neck portion. Five centimeters from the fundus there was a contracting ring 1 cm. in width and 2.5 cm. in circumference which constricted the gallbladder. The wall on section in the area distal to the constriction measured 8 mm.; proximally 4 mm. In the portion of the gallbladder distal to the adhesion band the mucosa was trabeculated; there was some cholesterolosis, and there were openings into Rokitansky-Aschoff sinuses which could be seen grossly on the cut surface of the wall of the gallbladder. The largest of these openings was 2 mm. in diameter. The sinuses grossly visible varied up to 3 mm. in diameter. In the proximal portion the mucosa appeared fairly well preserved. Three pigment type calculi, the largest 5 mm. in diameter were found within the lumen. Dark green tenacious bile filled the dilated Rokitansky-Aschoff sinuses; the bile found in the lumen of the gallbladder was golden yellow in color. Microscopic sections showed a marked chronic cholecystitis with marked thickening of the muscle layer and fibrosis of the subserosa. There was marked lymphocytic infiltration throughout the entire wall. There were many Rokitansky-Aschoff sinuses, some of which were very deep and large with retention of mucin. Some of the sinuses contained pigmented stones with mucin.

FIG. 4. CASE II. Note marked stenosis of gallbladder.

CASE III. A forty-six year old white female was admitted September 28, 1953, because of abdominal pain which first occurred three weeks prior to admission. This pain accompanied by nausea usually had its onset two to three hours after meals. There was an increase in the length and frequency of these attacks during the four days prior to admission. The patient had had several surgical procedures including appendectomy, thyroidectomy, oophorectomy and hysterectomy.

Physical Examination: The patient was a well nourished white female in acute distress. She had definite tenderness over the right upper quadrant of the abdomen with no palpable masses. No icterus was noted. Temperature, 99.6; pulse, 85; respirations, 20; blood pressure, 130/76.

Laboratory Findings: The urine was essentially negative. Erythrocyte count, 5,000,000; hemoglobin, 14.1 gm.; white blood cell count, 12,000; blood glucose, 103; urea nitrogen, 11; serum proteins—albumin 4.8, globulin 2.5; alkaline phosphatase, 10.1 (Bodansky units).

Fig. 5. Case III. Note stenosis with segmentation of gallbladder.

Roentgen Findings: Oral cholecystography on October 2, 1953, revealed excellent function. There was a constriction of the gallbladder at the junction of the upper and middle one third. This had the appearance of a septum, partially occluding the gallbladder. Careful study of the distal gallbladder shadow revealed very fine irregularities of the outline in the region of the fundus, suggestive of Rokitansky-Aschoff sinuses (Fig. 5). Emptying of post-fat meal was good in the proximal segment and poor in the distal segment. No calculi were demonstrated.

Operation: A cholecystectomy and choledochostomy were performed on October 7, 1953. A large thick-walled gallbladder was found. A match head size calculus was palpated in the cystic duct, and other small stones were palpated in the gallbladder. In the middle one third, a soft tissue diaphragm-like structure was palpated. The postoperative course was uneventful.

Surgical Pathology: The resected gallbladder measured 12.5 cm. by 4 cm. On section the lumen was distended with greenish black bile and contained numerous small bile calculi of the gravel type measuring up to 4 mm. The wall measured 2 mm. in thickness. Microscopic sections revealed the typical picture of chronic cholecystitis with several huge Rokitansky-Aschoff sinuses in the wall filled with soft gallstones. There was very marked hypertrophy of the musculature and considerable fibrosis of the subserous coat. The inflammatory pattern was entirely of the chronic type.

Case IV. A fifty-two year old, married white female was admitted November 21, 1953, because of pain in the back, left hip and ankle which followed an injury. The patient had slipped and fallen on October 17, 1953.

Physical Examination: The patient was not in acute pain. Her abdomen was soft and no masses were palpable. Her left ankle was tender and some edema was present.

Laboratory Findings: On three successive occasions the urine was negative. Erythrocyte count, 4,300,000; hemoglobin, 12.4 gm.; white blood cell count, 12,000; sedimentation rate, 31 mm./hour.

Fig. 6. Case IV. Stenosis with good visualization of bile duct radicles.

Roentgen Findings: Oral cholecystography on November 24, 1953, revealed excellent function. The gallbladder exhibited a constriction one inch distal to the cystic duct. The distal portion of the gallbladder contained negative density stones (Fig. 6). Because of the area of stenosis, the gallbladder was followed carefully after the fat meal. There was contraction of both segments, and as anticipated, the contrast medium entered multiple Rokitansky-Aschoff sinuses about the periphery of the distal segment.

Clinical Course: No surgery was performed and the patient was discharged on November 25, 1953.

CASE V. A forty-eight year old white female was admitted January 4, 1954, with the chief complaint of low back pain, and pain in the right thigh and leg of six months' duration.

Physical Examination: Tenderness was noted in the right upper quadrant of the abdomen. Neurologic examination was negative.

Laboratory Findings: Urine was negative. Erythrocyte count, 4,600,000; hemoglobin, 12.9 gm.; white blood cell count, 5,300.

FIG. 7. Case v. Note so-called "double gallbladder," on pre-fat meal roentgenogram.

FIG. 8. Case v. Distal segment only of gallbladder is visualized. It contains two calculi.

Roentgen Findings: Oral cholecystogram, January 7, 1954, revealed good function. A septum was noted at the junction of the proximal and middle one third. Two large negative density calculi were present distal to the septum (Fig. 7). Because of the gallbladder contour, the post-fat meal examination was carefully carried out with complete emptying of the gallbladder proximal to the septum. However, the distal portion retained its contrast medium and showed filling of quite typical Rokitansky-Aschoff sinuses throughout the entire circumference of the gallbladder wall distal to the septum (Fig. 8).

Clinical Course: The patient was discharged on January 10, 1954, and admitted to another hospital for surgery.

Operation: Cholecystectomy and appendectomy were performed January 25, 1954, without difficulty. The gallbladder contained two calculi 1 inch in diameter. The wall of the gallbladder was slightly thickened; no stones were palpated in the common duct.

Surgical Pathology: The gallbladder was 8.5 cm. long; the serosa was smooth. The muscle

wall was not remarkable, the mucosa was pale tan and finely granular. Two calculi 2 cm. in diameter were present. On section, there was formation of Rokitansky-Aschoff sinuses with inspissated substance in the sinuses surrounded by foreign body giant cells. A few chronic inflammatory cells were present in the wall.

DISCUSSION

We feel that stenosis of the proximal gallbladder is one of the most important etiologic factors in the cholecystographic demonstration of Rokitansky-Aschoff sinuses. In all of our cases (5) and in 10 to 15 cases by others, this process was obvious. In the presence of a distal septum, as in the phrygian cap which is seen frequently, the cholecystographic findings of Rokitansky-Aschoff sinuses were not found.

Pathologically, the stenosis or constriction was due to hyperplasia of all of the gallbladder wall structures and frequently contained Rokitansky-Aschoff sinuses with concrements within them. March[13] thought that the segment of stenosis was most likely congenital because of the presence of a fetal-type lobulated kidney in the same patient.

Roentgenographically, there was relatively good function of all cases reported; however, after the fat meal, there was better contraction of the gallbladder segment proximal to the stenosis. The Rokitansky-Aschoff sinuses were demonstrated more clearly and in greater numbers following the meal. Roentgenographic Rokitansky-Aschoff sinuses were demonstrated distal to the area of stenosis, which suggested the mechanism of increased intracystic pressure as a major etiologic factor in developing macroscopic herniations. Åkerlund and Rudhe[2] gave the following conditions for roentgen demonstration of diverticula (Rokitansky-Aschoff sinuses):

1. The gallbladder must be well filled with contrast medium of sufficient density.
2. The diverticula must be sufficiently large and numerous, and closely packed.
3. The diverticula must not contain any appreciable amount of retained bile components.

We should like to add a fourth condition, *i.e.*, a stenosis or segment of constriction is often present in the proximal portion of the gallbladder. In our third case, Rokitansky-Aschoff sinuses were seen on the roentgenograms only after very careful scrutiny, in the light of a definite stenosis of the proximal gallbladder. We feel that any cholecystograms which reveal a double gallbladder or stenosis should be suspected of also harboring Rokitansky-Aschoff sinuses, and these should be sought for most diligently. Careful studies after fat meal and, if necessary, re-examination of the patient to demonstrate this process should be made. We have used various projections and magnification studies with a 0.3 mm. focal spot tube. Due to the lengthy exposure required and the resultant motion, there was no definite advantage over standard roentgen methods.

FIG. 9. Gas filled gallbladder, with gas in the planes of its wall. Colon outlined with barium.

Åkerlund and Rudhe[2] noted the demonstration of Rokitansky-Aschoff sinuses in acute pneumocholecystitis in 7 out of 14 cases. A similar case was reported by one of us (G.J.C.[6]) in April, 1948, as acute gaseous cholecystitis (Fig. 9). In retrospect there is no doubt that the gas pattern is similar to the pattern of the contrast medium in the cholecystographic demonstration of Rokitansky-Aschoff sinuses (Fig. 9).

SUMMARY

1. The histologic features of Rokitansky-Aschoff sinuses have been given.

2. We have reviewed the available reported cases of cholecystographic demonstration of Rokitansky-Aschoff sinuses.

3. We have reported 5 cases, bringing the total number of reported cases to 20.

4. We have attempted to correlate the pathologic and roentgen findings.

5. A stenosis of the proximal gallbladder was present in 15 out of 20 cases. In all probability this is an etiologic factor increasing the intracystic pressure and giving rise to macroscopic Rokitansky-Aschoff sinuses which can be seen on cholecystography.

6. The presence of stenosis of the proximal segment of the gallbladder as seen in the cholecystogram should make the radiologist suspicious of probable Rokitansky-Aschoff sinuses distal to it.

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PHOTOTIMED ERECT COMPRESSION SPOT FILMS IN CHOLECYSTOGRAPHY

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A GALLBLADDER that cannot be cleared of overlying visceral gas presents a major problem in roentgenographic examination. So, too, does the gallbladder that contains multiple small calculi or in which small calculi are masked by the density of the contrast medium.[15]

Among the methods advocated for overcoming these difficulties, plain water enemas and the injection of pitressin have been used to clear the duodenum and hepatic flexure of gas.[9,15] Their disadvantages are the delay they cause and the undesirable side actions of pitressin.[20]

Sometimes the supine patient is rotated to the right before making an exposure. In the right lateral decubitus position, the gallbladder falls to the right side so that its shadow is separated from those of gas filled portions of the gastrointestinal tract. Small stones not previously discernible often become visible on films made with the patient so positioned.[19,27]

One of the oldest methods for avoiding overlying visceral gas in visualization of the gallbladder, and for detecting small stones, is to make the exposure with the patient erect.[1,2,3,5,6,11,12,13,29,31,32] Shadows of the gallbladder, and of stones it may contain, can then be projected free from disturbing shadows. Roentgenograms of normal gallbladders with the patient erect have shown layering of medium-laden bile, that in the fundus being more dense than that in the neck of the gallbladder.[11,13] When the patient is standing, small granules usually collect at the fundus where they can be visualized. Sometimes, and this is especially true of small nonopaque stones, they seem to float or can be seen as a layer near the top of the more dense bile.[1,6,12] Both calcified and cholesterol stones may have been difficult to see or, in fact, invisible when dispersed in dense bile when the patient was in the horizontal position.[1,6,12,13]

In the author's experience, the erect position and the use of graded compression[1,2,4,18,28,33] has resulted in the sharpest detail possible in making spot films of the gallbladder. Information is gained by these procedures that might otherwise be difficult to secure. The use of a phototimer,[8,14,16,22–26] of special design which is to be described elsewhere, has permitted automatic control of exposure time. Spot film roentgenography is one of the most valuable applications of photoelectric timers.[26] The timer ensures films of uniform density and frees the physician from the technical details of his equipment. Following is a report of a series of examinations which demonstrates the advantages of these techniques.

METHOD

Spot film roentgenographic examinations using a photoelectric timer, and with the patient erect, were made of 500 patients with suspected gallbladder disease.

The oral cholecystographic agents, iodoalphionic acid,*[7] iophenoxic acid,*[17,30,34] and iodopanoic acid[21] were used in doses of 3 grams. Each patient ate a meal high in fat content at noon on the day preceding examination. This provided the impetus for the gallbladder to empty and thus insure adequate concentration of the contrast medium.[7,10] The evening meal contained no fat. The medium was ingested following the meal. In the case of iophenoxic acid, ingestion was timed to precede the roentgenographic examination by about ten hours, the optimal interval for visualization with this medium.[30]

The gallbladder was first examined fluoroscopically. If it was not visible, a

* Supplied as priodax tablets 0.5 gm. (iodoalphionic acid) and teridax tablets 0.75 gm. (iophenoxic acid) by the Division of Clinical Research, Schering Corporation, Bloomfield, New Jersey.

second dose of medium was given and the examination repeated in 24 hours.

Posteroanterior exposures by standard Bucky technique were made first. Then the patient was placed in the erect position. The gallbladder was centered with the fluoroscope, compression applied, and a phototimed spot roentgenogram made. Some of these exposures were made without the use of a grid. In others a 30 inch aligned linear filter grid was used, and in the remainder a 36 inch aligned crisscross filter was employed. The time required for each patient was approximately five minutes.

RESULTS

In 440 of the 500 patients, the gallbladder could be freed of obscuring gas shadows by placing the patient in an erect position and using compression. The gallbladder shadow could not be cleared from that of the hepatic flexure, the small intestine, or the duodenum in 60 patients. Both the clinical and roentgen evidence in the latter indicated some inflammatory process with adhesions. There was no evidence of calculi, although the clinical picture resembled disease of the gallbladder. Upon operation in 40 of these 60 patients, inflammatory processes with adhesions were found.

Forty-six gallbladders could not be visualized, even after the administration of a second dose of contrast medium on at least two occasions, so that a pathologic condition was assumed.

The gallbladder could be visualized in 394 patients. In 259 of them, the organ appeared normal in function and contained no calculi. In 103 patients, the gallbladder contained calculi readily visible regardless of the technique used. The calcified stones in 19 of this group could be seen on a routine flat film of the abdomen.

The remaining 32 patients, in whom the gallbladders were visualized by the procedure described, had previously had one or more roentgenologic studies with no demonstration of calculi and an indication of apparently normal function. The results of these routine roentgenographic examinations with the patient prone are illustrated in Figure 1, *A*, *B* and *C*. Each of these 32 patients had had attacks of biliary colic, yet it was not until they were examined in the erect position, using the compression technique and making a phototimed spot film, that stones in the gallbladder became visible. Figure 2, *A*, *B* and *C* are simultaneous phototimed, erect, compression spot roentgenograms of the gallbladder of the same patient as in Figure 1, *A*, *B* and *C*. By this technique, calculi are clearly visible in a distinct dependent layer. Comparison of these roentgenograms illustrates the value of the phototimed spot film procedure described. At operation, calculi were found in all 32 of these patients.

Figure 3*B* is a phototimed compression spot roentgenogram in which a 14 mm. calculus is clearly visible. The patient in this

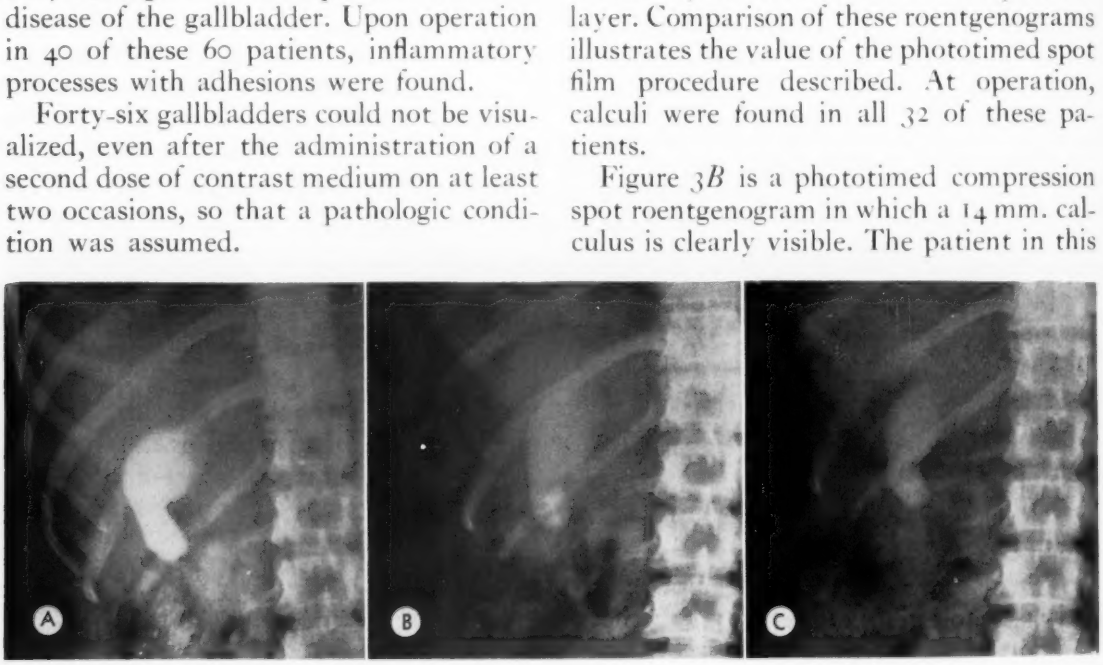

FIG. 1. (*A*, *B* and *C*) Cholecystograms made in prone posteroanterior projection. Calculi not visualized.

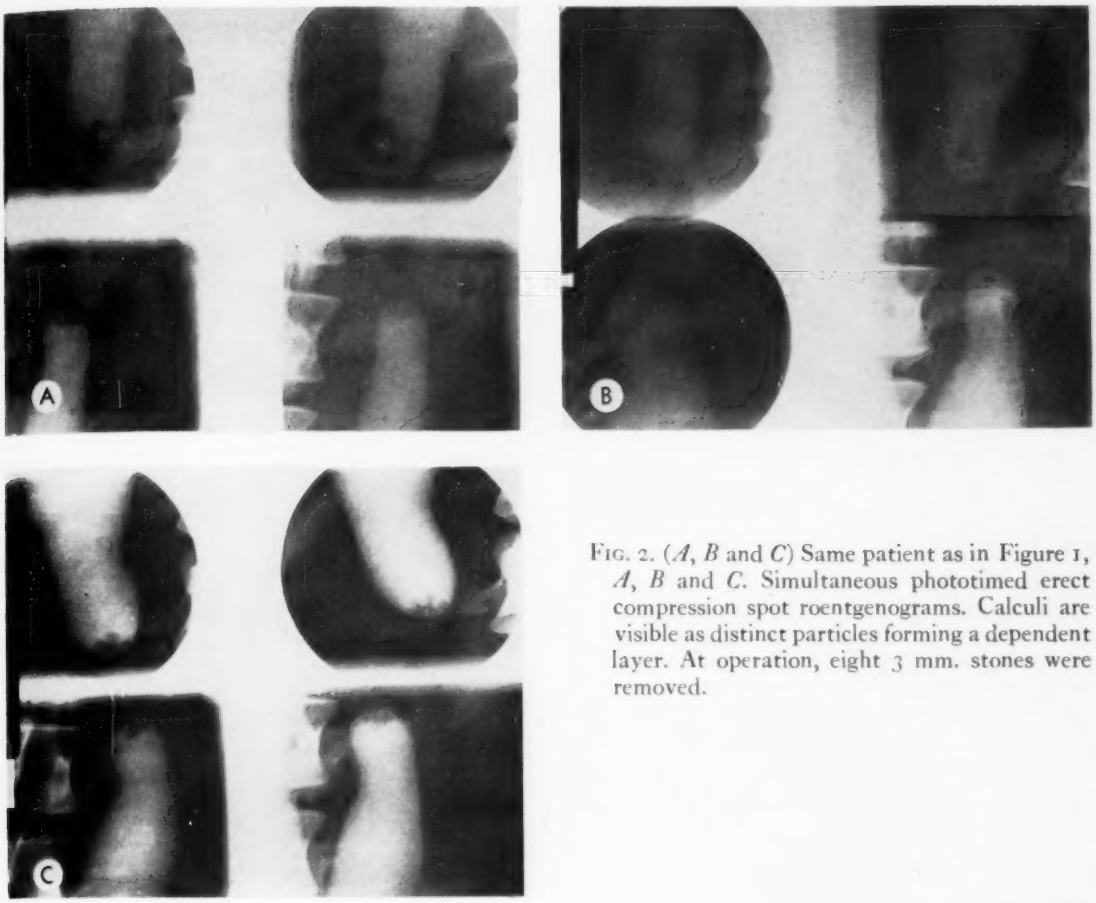

Fig. 2. (*A*, *B* and *C*) Same patient as in Figure 1, *A*, *B* and *C*. Simultaneous phototimed erect compression spot roentgenograms. Calculi are visible as distinct particles forming a dependent layer. At operation, eight 3 mm. stones were removed.

instance measured 33 cm. in thickness and weighed 200 pounds. When less compression was used, visualization became less distinct (Fig. 3*C*). The least distinct visualization was obtained in the posteroanterior projection with the patient prone (Fig. 3*A*).

The spot roentgenograms taken by the phototimed method were uniformly exposed as can be seen in Figure 2, *A*, *B* and *C*. Each film contains serial spot exposures made without grids, and with linear or crisscross grids. All films were compared on a densitometer and found to be within 5 per cent of the average density. It will be noted that the conventional longitudinal grid approximately doubles the radiation density required to produce the same shadow over that without a grid, while the crisscross grid quadruples it.

Comparing the results with the three contrast media used, it was found that small calculi were less likely to be obscured by a medium that produces a moderately dense shadow than by one that produces a very dense shadow. In these patients, iophenoxic acid proved to be preferable to the other media used for gallbladder visualization.

DISCUSSION

A normally functioning gallbladder may concentrate cholecystographic media to such an extent that small calculi are obscured. In the horizontal position, small stones in the gallbladder may be covered with iodine-containing bile of such density that they will not visualize.[15] Generally, a stone larger than 15 mm. in diameter will displace sufficient medium to visualize with the patient in any of the positions usually used in cholecystography. Stones between

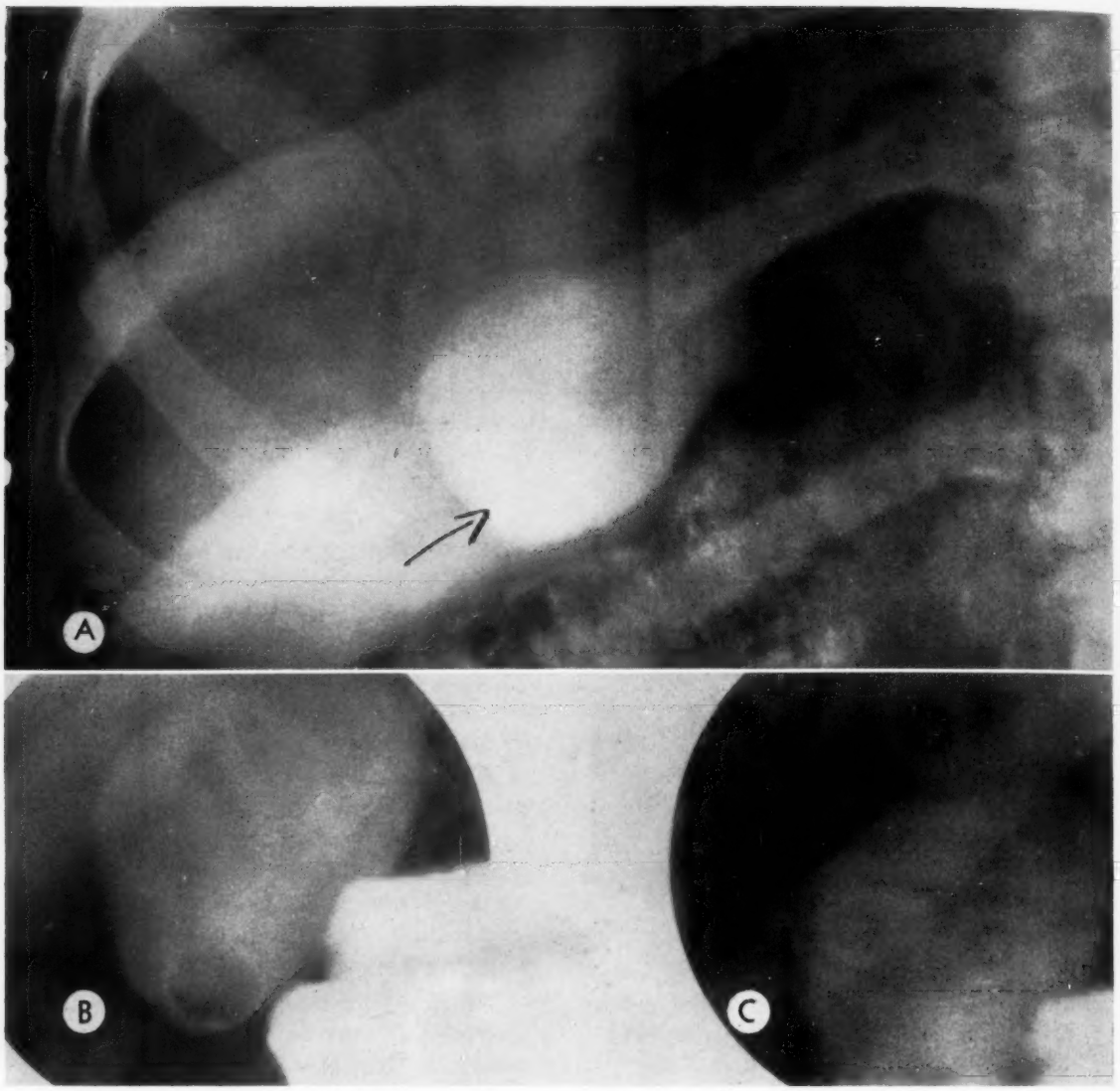

FIG. 3. (*A*) Prone posteroanterior projection resulting in least distinct visualization. Patient measured 33 cm. in thickness. Weight 200 pounds. (*B*) Same patient as *A*. Compression spot roentgenogram with 14 mm. calculus clearly outlined. (*C*) Same patient as *A* and *B*. Spot roentgenogram for which less compression was used, so that visualization was less distinct.

10 and 15 mm. will border on visualization, while stones under 10 mm. in diameter may be missed. Although not usually visualized in posteroanterior projections with the patient prone, these small stones can be demonstrated readily in spot films with the patient erect. Their visualization is assisted by a compression technique which places the radiolucent stones on the margin of the gallbladder with the thinnest possible layer of contrast medium superimposed.

If the gallbladder is visualized fluoroscopically and roentgenographic studies made with graded compression, the following advantages may be obtained:

1. The gallbladder shadow can be freed from those of overlying visceral gas and of bony structures such as the spinal column.

2. The roentgenographic thickness of the gallbladder can be decreased so that sharper detail with less fogging of the film from secondary radiation is achieved.

3. The diameter of the gallbladder can be reduced by compression to a thickness to

permit visualization of small, radiolucent calculi.

The major disadvantage to spot filming in the past has been exposure control. In the dark room it is difficult, as the patient is rotated, to measure patient thickness. Even with accurate measurements, it is difficult to judge the effect of graded compression in reducing density. Phototiming overcomes these difficulties and controls the exposure time automatically. With its use, uniform predictable roentgenograms can be produced.

SUMMARY

Five hundred patients suspected of having gallbladder disease were examined roentgenographically after the ingestion of iodoalphionic acid, iophenoxic acid, or iodopanoic acid. Phototimed spot exposures employing compression, and with the patient erect, rapidly provided roentgenograms of uniform diagnostic quality. With this technique, the shadow of the gallbladder in 440 patients could be projected free from those of overlying visceral structures containing gas. In 40 of the 60 patients where the shadow of the gallbladder could not be projected separately, inflammatory processes with adhesions were found at operation. No gallbladder disease was present in 259 patients. Visualization failed in 46 patients, even after repeat doses of contrast medium. Calculi were present in 103 patients and visible on both posteroanterior projection roentgenograms and those made with the patient erect. In 32 patients, calculi were seen for the first time on the erect, compression spot film, whereas they had not been visible in posteroanterior projection with the patient prone. This finding demonstrates the value of phototimed spot films made with the patient erect and employing compression.

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AN EVALUATION OF TWO RECENTLY INTRODUCED ORAL CHOLECYSTOGRAPHIC MEDIA

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TWO recently introduced cholecystographic media form the basis of this study. The first is iopanoic acid (telepaque) and the second iophenoxic acid (teridax).* Iopanoic acid [β(3-amino-2,4,6 triiodophenyl) α-ethylpropionic acid] containing 66.68 per cent iodine was introduced in 1951. It is rapidly absorbed from the intestinal tract and appears in the bile within two hours after administration. It is excreted primarily in the intestinal tract. Iophenoxic acid is excreted primarily by the kidneys, and therefore no residue is found in the intestinal tract. Earlier drugs gave rise to a number of undesirable side reactions from which the two under discussion are relatively free.[2,3,5,6]

Iopanoic acid has been shown to give a more dense shadow.[3,4] It is debatable whether this aids or retards the demonstration of gallbladder disease. Other investigators feel that the presence of unabsorbed or excreted iopanoic acid in the colon is a drawback because it interferes with visualization.[4] Iophenoxic acid (teridax) is free from this criticism. The bile ducts are often visualized during the course of the examination with telepaque, but this occurs only in the presence of a relatively normally functioning gallbladder.[7] Information about the biliary tree is most frequently desired in the presence of a calculous or a nonvisualized gallbladder, hence, telepaque is of extremely limited value in this situation. Information about the biliary tree can best be obtained by use of the newer intravenous cholecystographic media.

METHOD

The clinical trial was set up to determine the relative value of teridax and telepaque. The hypothesis to be evaluated was that no difference exists between the two chemicals. A random sample was obtained by following the tables of random numbers given by Fisher.[1] Following the table each consecutive out-patient referred for gallbladder examination was assigned a number. The odd numbered patients were given 4 tablets (0.75 gm. per tablet or 3 gm. per patient) of teridax; the even numbered patients were given 6 tablets (0.5 gm. per tablet or 3 gm. per patient) of telepaque. All of the patients were adults and for the sake of uniformity the dose did not vary with the weight of the patient. The doses given are those recommended by the manufacturer.

Roentgenograms of the gallbladder were obtained after an interval of from 12 to 16 hours. If the gallbladder was not visualized, the patient was given another dose of teridax, or telepaque (identical with the first dose) and the films were obtained after another interval of 12 to 16 hours. The physician who examined the films did not know in any case which chemical the patient had received. The results of the test were recorded in one of four categories: (1) stones, (2) good visualization, no stones, (3) poor visualization, no stones, and (4) no visualization. The results are shown in Table 1.

Untoward reactions from either contrast medium were not recorded. They were minor and infrequent in both series and in

TABLE I

	Stones	No Visualization	Diminished Concentration	Good Concentration	Total Cases
Teridax	9	7	7	11	34
Telepaque	6	8	4	14	32

* Iophenoxic acid (teridax) was contributed by the Schering Corporation, Bloomfield, New Jersey.

no instance did they interfere with the performance of the examination. These minor reactions have been reported in detail by other authors.[4]

Since the purpose of the gallbladder examination is to determine the presence of disease, that drug which will best determine this is most valuable. Fortunately, the end point, as far as clinical tests go, is fairly well defined. Either the gallbladder has stones or it does not have stones. Either the gallbladder is visualized, or it is not visualized. In the first instance it is generally agreed that the presence of stones indicates stasis or infection, or both. (No patients with detectable hemolytic disease were included in the study.) With a cooperative patient who takes the medication as directed and in the absence of vomiting or diarrhea, failure of gallbladder visualization also indicates disease in the majority of instances.[8,9]

Variations in the density of the contrast material in the gallbladder are difficult to evaluate. In the first place, there is individual variation in the rapidity and effectiveness of absorption from the gastrointestinal tract. Secondly, technical factors in the production of the roentgenogram, as well as the thickness of the patient, will result in variations of the density of the medium seen in the gallbladder. For these reasons an assessment of the intensity of concentration of medium seen in the gallbladder is of dubious value.

RESULTS

This is a test of gallbladder disease and the single most important indication is the presence of calculi. In this study calculi were found in 9 patients out of 34 examined with teridax, and 6 patients in 32 examined with telepaque ($X^2 = 3.22$ and P is greater than 0.05). There is, therefore, no evidence that the observed difference is other than that which might be obtained by random sampling. The difference is not significant in the two series if the demonstration of calculi is taken as the criteria for evaluation. It follows that this study does not show a superiority of one drug over the other in the delineation of gallbladder disease.

Table II

PATIENTS OVER SIXTY YEARS OF AGE

	No Stones	Stones	Total
Teridax	15	4	19
Telepaque	12	1	13

To test for the randomness of the two samples it seemed expedient to divide them into two age groups—over sixty years, and under. The incidence of calculi may increase in the older age group, and if a large number of patients over sixty were to be found in one or the other series, despite the method of random sampling, this would unduly weigh the results in favor of that drug. Table II shows the distribution of patients over sixty years of age according to the type of medication ($X^2 = 2.76$, P greater than 0.05). Hence, there is no evidence that this is not a random sample.

A double dose of the medium was given to 9 patients who had initially received teridax, and to 10 patients who had initially received telepaque. No visualization was encountered in 7 patients after teridax, and in 8 patients after telepaque. Thus, in all other respects no significant difference was found between the two contrast materials as regards their roentgenographic diagnostic value.

CONCLUSION

1. The reliability of teridax and telepaque in the detection of gallbladder disease in two groups of out-patients was tested.

2. The grouping of the patients into those receiving teridax or telepaque was carried out on a random basis. The assessment of the value of one or the other drugs was carried out blindly. The examiner did not know which the patient had received.

3. With respect to its value in the demonstration of gallbladder disease, no significant difference between teridax and telepaque was found in this series.

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A NEW HEPATOLIENOGRAPHIC AGENT: TIN OXIDE

PRELIMINARY REPORT

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FOR roentgen visualization of certain organs, the constituent cells must take up radiopaque material. The phagocytic reticuloendothelial cells, found in great abundance in liver, spleen, and placenta, have the ability to ingest huge numbers of colloidal particles. When these particles are of suitable radiopacity, liver, spleen, and placenta may be well demonstrated. The colloidal compound must be introduced into the blood stream without immediate or ultimate harm to the organism. Substances previously investigated experimentally and clinically have not been found satisfactory. The iodinated fatty compounds have had immediate toxicity. The commercially available heavy metal compound, thorium dioxide, is radioactive. It is productive of tissue fibrosis and is suspected of predisposing to neoplasia.[4]

Colloidal tin oxide is an ideal hepatolienographic agent. Tin has excellent radiopacity in the kilovoltage range of clinical usage. It is established that long retained tin oxide in the human and in the animal is innocuous. Suspensions of tin oxide of an individual particle size not exceeding two microns were found to be well tolerated when introduced into the blood stream of experimental animals. Rapid intravenous injections in twenty-six separate instances were without event in 9 rabbits and 4 dogs. The colloidal tin oxide was usually given in doses of about 350 mg. of tin per kg. of body weight, at the rate of 15 to 144 mg. of tin per kg. of body weight per minute. The upper limit of safe dosage of colloidal tin oxide is not yet known, but one rabbit received 1,578 mg. of tin per kg. as a total amount from four divided injections without reaction or obvious harm.

Uniformly excellent and extremely dense shadows of normal liver and spleen were observed following doses of about 500 to 700 mg. tin per kg. of body weight, and in some cases in doses less than this (Fig. 1, *A* and *B*). The placentae were visualized in one attempt in a pregnant rabbit by similar dosage. In 2 rabbits, a "spotty" liver shadow was observed. Postmortem examination revealed extensive, diffuse involvement of the liver parenchyma by the lesions of coccidia in these 2 animals.

The optimum dosage for constant reproducible visualization of liver and spleen is not yet established but probably lies between 400 and 700 mg. of tin per kg. body weight. Liver and spleen shadows appear to reach their maximum density about four to six hours after intravenous injection. Visible fading of the shadows has not been observed during the present observation period. Gross anatomic and histologic studies of rabbit livers and spleen, which had contained the tin oxide for as long as 30 days, and of the dog for 100 days, did not reveal any evidence of cell damage, proliferative reaction, or fibrosis. The tin oxide could be demonstrated intracellularly in liver and spleen. A minute amount was in lung phagocytes.

The clinical applications of colloidal tin oxide can be envisioned if further studies now in progress confirm its total lack of immediate toxicity. Chronic toxicity should not be expected since tin oxide has been present in the lungs of man for as long as forty years without causing physiologic embarassment or anatomic damage.[3] Any slow breakdown of tin oxide into ionic tin would not be expected to be harmful, for the human body is exposed to considerable quantities of tin from canned food ingestion and no toxicity results.[2] The nonreactivity of tin oxide with the tissues minimizes the theoretical objection of long retention in the body, which this type of contrast medium will naturally have. Unequivocal proof exists that widespread deposition of tin oxide in lung lymphatics

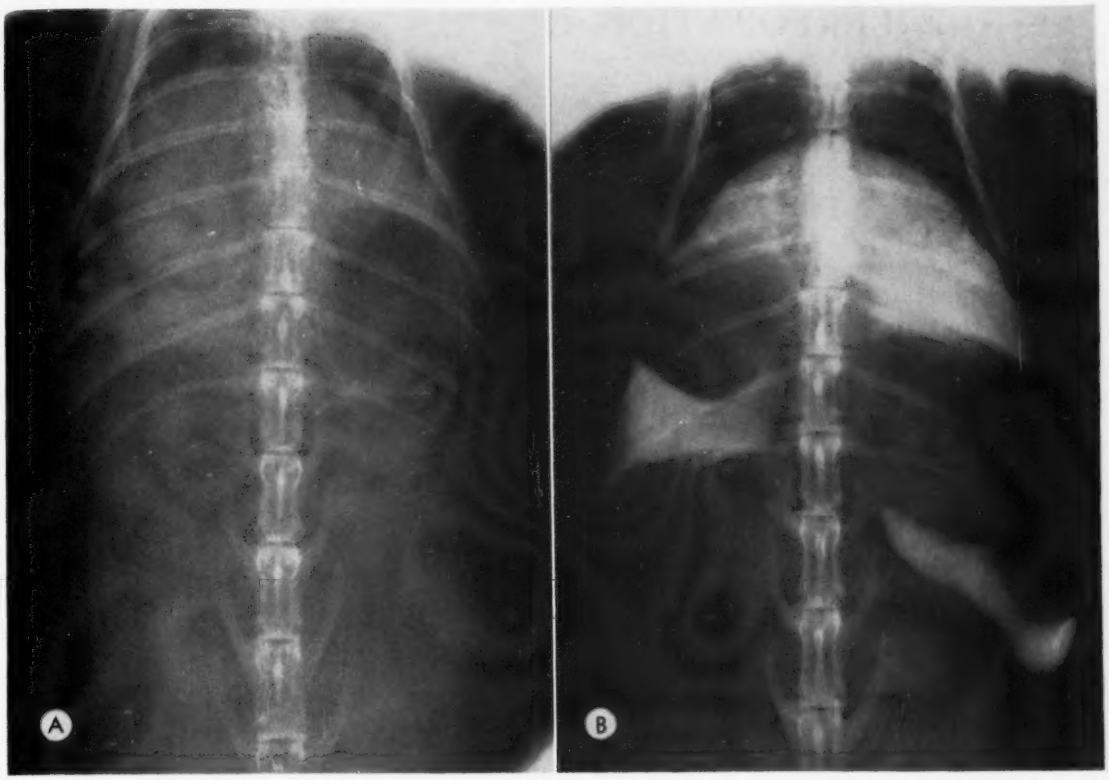

FIG. 1. Roentgenogram of 3.8 kg. rabbit, demonstrating excellent liver and spleen shadows obtained by a new hepatolienographic agent, colloidal tin oxide. (*A*) Pre-injection. (*B*) Following intravenous injection of 748 mg. Sn as colloidal tin oxide per kg. of body weight. Identical technical factors utilized for each roentgenogram (60 kv., peak, 200 ma., 1/20 second, Bucky diaphragm, 30 inch tube-film distance, 3 mm. aluminum tube filtration added).

is not accompanied by any special fibrous reaction.[1]

Further studies are planned or are in progress to investigate other usages of tin oxide as a contrast agent.

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PERFORATION OF THE RECTUM*

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A REVIEW of the literature fails to reveal a detailed discussion of the roentgenologic aids in the diagnosis of perforation of the rectum. In several papers in the surgical literature, mention is made of the demonstration of free air in the peritoneal cavity following intraperitoneal perforation of the rectum. Only brief mention is made of the diagnosis of extraperitoneal perforation. Thus, Bernstein *et al.*[2] describe a case of emphysema of the leg following perforation of the rectum. In their interesting paper they state that, "an area of gas was seen in the pelvis in an area which seemed to be lateral to the lumen of the rectum." L. P. Thomas[9] states that air may be demonstrated lateral to the rectal wall.

The purpose of this presentation is to discuss and demonstrate air lateral to the rectal wall as an aid in the diagnosis of rectal perforation.

PRELIMINARY CONSIDERATIONS

Perforation of the rectum may occur as the result of: (1) External trauma: (a) bullet and stab wounds (Fig. 2); (b) crushing injuries (Fig. 3); (2) Internal trauma: (a) perforation during proctoscopy; (b) perforation by enema; (c) perforation by impalement; (3) Nontraumatic: (a) rupture of pararectal abscess into rectum (Fig. 1); (b) spontaneous—cause unknown.

Reports in the literature stress the seriousness of the injury and the need for early diagnosis. The mortality rate varies from 20 to 50 per cent and all observers agree that the rate increases with the delay in diagnosis and early definitive surgery.

The immediate recognition of perforation of the rectum is not always obvious. Shock, bleeding and even pain may be absent. An actual demonstration of a rectal perforation is, of course, the best and quickest means of early diagnosis. Its demonstration demands immediate definitive surgery.

In the absence of a demonstration of a rectal perforation, the roentgenogram may be a great aid and a film of the abdomen should be made in all cases suspicous of rectal perforation.

FIG. 1. Air in pararectal tissues, the result of spontaneous rupture of rectum due to abscess.

ANATOMICAL CONSIDERATIONS

The upper two thirds of the rectum have a partial covering of peritoneum in the front and at the sides and, later on, in the front only. The lower one third has no peritoneal covering. When the mesocolon ceases at the end of the pelvic colon, its two layers separate and leave the back of the rectum free of peritoneum. Soon the membrane quits its sides also and is then found on the front only. Thus, almost the entire rectum lies behind or beneath the pelvic peritoneum and failure to demonstrate free air in the peritoneal cavity does not exclude rectal perforation in the entire lower third and the posterolateral surfaces of the upper two thirds of the rectum.

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Fig. 2. Perforation of rectum due to war wound. Characteristic air streaks are seen bilaterally.

A perforation of the rectum may be either intraperitoneal, extraperitoneal or combined. An intraperitoneal perforation of the rectum is diagnosed by a demonstration of free air in the peritoneum and is well known to all. Diagnosis of extraperitoneal rupture of the rectum is made by a demonstration of air in the extraperitoneal tissues surrounding the rectum. The air in this space is due to an escape of air from the rectum and/or the formation of gas due to colon bacillus infection. Its demonstration with, or without, the presence of free air is positive indication of a rectal perforation and may be of some help in localizing the portion of the rectum involved.

ROENTGENOLOGIC CONSIDERATIONS

The air within the pararectal tissues is distributed in a linear fashion in the periphery of the pelvis lateral to the rectum. This linear collection of gas tends to be convex outward and its distribution appears to follow the contour of the pelvic wall, coursing towards the midline as it approaches the anus. It may be unilateral or bilateral and, more often than not, is

Fig. 3. Perforation of rectum due to severe crushing injury.

Fig. 4. There is a characteristic pararectal air streak, but a perforation was not found. This was a war wound contaminated by feces. The air was probably formed by gas producing organisms.

unilateral. This linear streak of air may, on closer analysis, be seen to consist of minute bubbles of air of varying sizes. This collection of bubbles of air does not correspond to any known normal anatomic part. It is more radiolucent than gas within the rectum. Its appearance does not change with change in position and, unlike gas in the colon, has the same appearance from hour to hour. The extent of this linear shadow may increase with time, probably because of colon bacillus infection.

This appearance is very characteristic and diagnosic. In the author's experience, which included many war wounds, a mistake was made once in a patient who was shot in the buttocks and the wound became contaminated with fecal material. This produced a linear streak similar to the one described above (Fig. 4).

SUMMARY

1. It is of great importance to recognize and accurately diagnose rectal perforation.

2. The diagnosis may be made roentgenologically by the demonstration of free air. However, a large part of the rectum is extraperitoneal. Demonstration of a characteristic pararectal linear streak of air bubbles is diagnostic of extraperitoneal perforation.

3. The roentgenologic diagnosis is easy if one looks for this shadow.

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WATER INTOXICATION

REPORT OF A FATALITY FOLLOWING A BARIUM ENEMA

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THE potential and real dangers of tap water enemas administered to patients with congenital megacolon were reported approximately one year ago by Richards and Hiatt.[3] Steinbach, *et al.*[4] also reported on the danger of producing water intoxication with hypotonic enemas in cases of megacolon. In 1952, Baskin, Keith and Scribner[1] reviewed the literature on water intoxication. They noted several reported cases of water intoxication following proctoclysis of hypotonic solutions. Recently, Hill[2] reported a patient with a normal colon who was a victim of hypervolemia and hypoelectrolytemia as a direct result of the administration of hypotonic enemas. He pointed out the danger of ordering the administration of tap water enemas until clear as a part of the routine procedure for preparing the intestine for fluoroscopic and roentgenographic examinations.

Approximately concurrent with the appearance of Hill's case report, one of our patients expired from hypervolemia and hypoelectrolytemia following a barium enema for the roentgen examination of the large bowel. To our knowledge, this is the first reported fatality in a normal child from water intoxication following a barium enema.

REPORT ON A CASE

J. H., a three year old white male, entered the hospital on November 24, 1954, for studies in regard to a persistent and progressive constipation since birth.

The child had been a patient in this hospital on five different occasions in the previous twenty one months. His first admission had been for a Salmonella diarrhea and dehydration from which he made a rapid and uneventful recovery. The next three admissions were for surgical procedures which included two squint operations and a tonsillectomy. One month prior to the present entry, he had been admitted because of rectal fecal impaction. This impaction was easily removed by enemas. At the time of this admission, the mother related that the child had always been somewhat constipated, but it was not until the last two years that he had any particular distress. Three or four times during the last year, she had noted occasional blood streaking on his stools. She frequently had found it necessary to use soap suppositories to cause bowel movements. The plan of the present admission was to cleanse the bowel by laxatives and enemas in order to perform proctoscopy and to do a barium enema. Aside from the previous hospital admissions, the past history was essentially negative, except for the fact that the patient had not yet begun to talk.

The physical examination at the time of admission revealed a small, strong and uncooperative three year old white male who did not talk. His tympanic membranes were retracted and slightly red. The heart and lungs were clear. The abdominal wall was held rigid voluntarily. The extremities were thin. Routine laboratory work on the day of admission revealed urine specific gravity 1.020 with albumin, sugar, acetone and sediment being within normal limits. The white blood cell count was 8,350; hemoglobin, 13.1 grams. The differential count was essentially normal.

He was started on a low residue diet and was given one ounce of phosphate of soda at bedtime. The night of admission he was also given a soapsuds enema and a one ounce vegetable oil retention enema. Following the laxative and enemas, he had several large, liquid, foul-smelling green stools. The following morning, a repeat urinalysis revealed a specific gravity of 1.030 with a trace of albumin, 3-plus acetone, 4 to 6 white blood cells, 2 to 3 red blood cells, and an occasional granular cast. During the day, his intake was poor and he vomited several times. Another dose of phosphate of soda was administered. That evening, he was given 600 cc. of saline solution as an enema until the return was clear. The following morning, he drank a little juice for breakfast and seemed quite well. He was ambulatory about the ward

and in no distress. He had a proctoscopic examination which was negative and went to the radiologic department at noon. Barium enema examination revealed a normal large bowel. Approximately an hour after the barium enema, he was given a tap water enema, at which time he expelled a relatively large amount of soft stool with many particles of barium. Fifteen minutes after this tap water enema, he vomited and then became flushed and lethargic. Examination at this time revealed a flushed child lying quietly in bed but responding to stimuli by screaming and becoming rigid. His right limbs were much more rigid than the left. The blood pressure was 120/80 and the pulse rate 100. Respirations were irregular at a rate of 20. The pupils were both widely dilated and did not react to light. The fundi appeared normal. The jaws were tightly clamped. The heart and lungs were essentially normal. The abdomen was flat and soft. The skin and mucous membranes appeared only slightly dehydrated. It was thought that he was suffering from central nervous system disease and was consequently taken immediately to the treatment room where a lumbar puncture was done. The fluid was clear. The cell count was 2 white blood cells and 1 red blood cell and the protein 31.4 mg. per cent. During the procedure, the patient suddenly became much worse. He became limp and cyanotic and began to froth at the mouth. An infusion of 5 per cent dextrose in normal saline was immediately started. Other emergency measures included oxygen, suction, intravenous and intracardiac adrenalin, intravenous hydrocortisone and artificial respiration. All procedures were to no avail. The patient did not respond and was pronounced dead approximately one hour after onset of his comatose state. Gross and microscopic postmortem examination revealed only pulmonary and cerebral congestion and a bicuspid aortic valve. The immediate postmortem serum chloride was 83 meq/l (normal 98–106). It was felt that the cause of death in this case was hypervolemia and hypoelectrolytemia.

DISCUSSION

The similarity between the clinical features of this case and of that reported by Hill[2] is striking. The poor intake and the vomiting preceding the barium enema in our patient undoubtedly contributed to his susceptibility to water intoxication from the absorption of large amounts of hypotonic enema solution. It appears, however, that the barium enema and the following tap water enema were the major factors in precipitating his fatal electrolyte disturbance. We agree with Hill[2] that isotonic saline solution should be used for routine enemas and that the barium used for barium enema studies should be suspended in isotonic saline.

SUMMARY

A case of a fatality due to water intoxication following a barium enema for roentgenographic examination of the large bowel is reported. The importance of using isotonic saline solutions for routine enemas is emphasized.

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THE ROENTGEN FEATURES OF FIBROUS DYSPLASIA OF THE SKULL AND FACIAL BONES*

A CRITICAL ANALYSIS OF THIRTY-NINE PATHOLOGICALLY PROVED CASES

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IT IS the purpose of this paper to demonstrate clearly the multiplicity of the roentgen features of fibrous dysplasia as it involves the skull and facial bones; to propose a workable classification of these lesions; to emphasize the importance of eliminating older nomenclature, and to show that roentgen examination often has a high degree of diagnostic specificity. To my knowledge, no series of cases this large, all verified by microscopic study and roentgen examination, has thus far been published.

Fibrous dysplasia is a disease of one or many bones in which basically fibrous tissue lesions are found. This fibrous tissue has the property of expansion and distorts the involved bone and the variable potentiality to form bone directly (metaplasia).

When this study was undertaken, criteria were established so that all cases could be uniformly evaluated. Every case must have (1) satisfactory roentgenograms of the skull and facial bones, (2) an adequate clinical record, and (3) microscopic confirmation. The cases were collected from cooperative individuals and institutions in various localities throughout the United States.††

It is difficult to review the literature on this subject. The "Quarterly Cumulative Index Medicus" does separate fibrous dysplasia as an entity; however, the disease is listed among several other categories which include osteitis fibrosa, osteoma, etc., because various authors still prefer this terminology. Furthermore, in the literature prior to 1938, when Lichtenstein[13] introduced the term, "fibrous dysplasia," confusing nomenclature existed which is still being used. Many of these terms indicate different diseases; many the same disease. The following terminology is frequently used:

Generalized osteitis fibrosa cystica not due to hyperparathyroidism
Osteitis fibrosa in multiple foci
Leontiasis ossea
Osteitis fibrosa
Focal osteitis fibrosa
Unilateral von Recklinghausen's disease
Unilateral polyostotic osteitis fibrosa
Precocious puberty and bone brittleness
Osteitis fibrosa cystica congenita
Osteitis fibrosa cystica
Osteitis fibrosa disseminata
Osteitis fibrosa cystica localisata
Osteitis fibrosa cystica generalisata
Osteodystrophia fibrosa cystica generalisata limited to one side of the body
Osteodystrophia fibrosa
Osteodystrophia fibrosa unilateralis
Juvenile Paget's disease
Von Recklinghausen's neurofibromatosis
Osteofibroma

†† Nine cases from the United States Naval Hospital, National Naval Medical Center, Bethesda, Maryland; 9 cases from the Walter Reed Army Medical Center, Washington, D. C.; 7 cases from the Armed Forces Institute of Pathology, Washington, D. C.; 4 cases from Dr. T. F. Hilbish of the National Institute of Health, Bethesda, Maryland; 2 cases from Dr. Ralph Caulk, Episcopal Eye, Ear, Nose and Throat Hospital, Washington, D. C.; 2 cases from Dr. Don C. Weir, St. Louis City Hospital, St. Louis, Missouri; 2 cases from Drs. Stanley Rogoff and Lucy Squire, Strong Memorial Hospital, University of Rochester Medical Center, Rochester, New York; 1 case from Dr. Owen Doyle, Lt. (MC) USNR, United States Naval Dispensary, Washington, D. C.; 1 case from Dr. John Hatch, Capt., MC, USN, United States Naval Hospital, Chelsea, Massachusetts; 1 case from Dr. John McClintock, Albany Hospital, Albany, New York; 1 case from Dr. Paul Popovich, United States Veterans Hospital, Jefferson Barracks, Missouri.

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Fibro-osteoma
Maladie de Charles Ruppe
Hyperostosis
Fibrous osteodystrophy
Regional fibrocystic disease
Disseminated fibrous osteitis
Ossifying fibroma
Jaffe-Lichtenstein disease

In any event, the elimination of these terms when referring to fibrous dysplasia would serve a useful purpose since fibrous dysplasia is a well documented, accepted clinical entity, and no other terminology more adequately expresses the disease.

The academic evolution of this disease has been slow. Before and during the 1920's, the term, "osteitis fibrosa cystica," was the most commonly used term to designate the fibrocystic lesion of bones. In 1926, Mandl[20] successfully operated upon and removed a parathyroid adenoma from a patient suffering from this disease. The era of hyperparathyroidism followed, and surgery was performed on many patients with "fibrocystic" bone changes. All of the patients operated upon did not materially benefit, since all of the patients explored did not have parathyroid adenomas or hyperplasia. Furthermore, the operative failures did not have the serum and urine calcium and inorganic phosphorus imbalance. Gradually, the limitations of the parathyroidectomy then became evident. Patients with bony fibrocystic disease who do not have parathyroid adenomas fall into a large category, the bulk of which is fibrous dysplasia. Therefore, the large group of osteitis fibrosa cystica has been defined, for the most part, into two entities,—hyperparathyroidism and fibrous dysplasia.

In an attempt to classify fibrous dysplasia, it seems wise to follow the present concepts of the process:

(1) This disease may involve any bone; however, when one bone is involved, the term, "monostotic," is used as a prefix.

(2) When more than one bone is involved, the term, "polyostotic," is used.

(3) The third form of fibrous dysplasia exists in association with hyperpigmentation of the skin, precocious menstruation in females, and early skeletal maturation. This is known as Albright's disease.[1,2,21]

PATHOLOGY

The gross pathologic features were recorded only a few times in this series. In 6 cases, the entire lesion was resected and thoroughly studied. In some cases, the lesions were inspected surgically, but were not entirely resected. In these patients, when indicated, the lesion was altered surgically so the bone contour resembled the uninvolved side. Other cases were biopsied through a small incision to establish the diagnosis, and gross visual study of the lesion was not made. One case presented many peripheral bone lesions in addition to lesions of the skull and facial bones. In this case, the biopsy was performed on a more accessible peripheral bone.

The gross findings show, in the studied cases, enlargement of the involved bone. The lesion may be white in color, and the consistency ranges from firmness to rubbery softness. Some areas are gritty because of the bone spicules in the fibrous tissue. The line of demarcation between disease and normalcy is not always definable. The involved area has a thinned cortex, and in the skull, a thinned outer table which, at times, may not be intact. Less frequently, the inner table also may be thinned.

The microscopic findings present a primary component of connective tissue.[25] These cells, which are fibroblasts, can arrange themselves in whorls. In some areas this connective tissue may be cellular, and in other areas, the prominent feature may be fibroblastic differentiation resulting in mature connective tissue, rich in collagen. Bone trabeculae are found throughout the fibrous tissue. These vary from small, immature, poorly calcified areas of osteoid to large well developed bony regions that reveal Howship's lacunae.[13] Some specimens will show the bony trabeculae to be scattered in a haphazard fashion, and other specimens demonstrate the irregular bony spicules to be uniformly distributed

throughout the lesion (Fig. 2). Large areas may show no evidence of bony spicules, while in the same specimen, there may be dense bony aggregations with less fibrous tissue (Fig. 3).

The vascularity of these lesions is variable. For the most part, vascular channels are not striking. It has been observed that where the deposition of bone proceeds more rapidly, the more abundant the vascularity; and, to the contrary, the less vascular areas will have more collagen and less bone.[13] The operative reports indicate some lesions have an excessive amount of vascularity, and in one case, the diagnosis of an arteriovenous fistula was suspected. In this case, the microscopic findings plus cerebral angiography did not confirm these suspicions.

As a rule, there is a paucity of osteoclasts. They are multinucleated and distributed throughout the connective tissue and on the margins of the trabeculae. Special stains for fat and myelin do not reveal these structures. No evidence of primary osseous malignancy was observed; however, this observation was recorded in 2 cases reported by Coley and Stewart,[5] and no evidence of cartilage was found in these specimens.

In the differential diagnosis from the pathologic side, one should mention nonosteogenic fibroma of bone. This lesion is composed of connective tissue, the cells of which are arranged in whorls, but exhibit no tendency towards osseous metaplasia. Furthermore, the lesion seems limited to children and adolescents. It is important to note that it appears to be confined entirely to the peripheral bones and the limbs but does not involve the skull.[25] The lesion is usually small in size, from one to several centimeters, and occurs near the epiphyseal plate within the cortex of the bone. Microscopically, the whorled connective tissue without evidence of osseous trabeculae is prominent. There may be multinucleated giant cells which occasionally contain lipoid.[14] These lesions have been referred to as cortical defects[4] and by some,[27] because of the lipoid material, erroneously labeled as lipoid gránulomatosis. This point will be discussed in more detail under pathogenesis.

Schlumberger[25] points out that the so-called nonosteogenic fibroma will reveal trabeculae of new bone in occasional areas. Since the connective tissue pattern is indistinguishable, he considers nonosteogenic fibroma to be a variant of fibrous dysplasia.

Differentiation of fibrous dysplasia from the so-called osteogenic fibromas and ossifying fibromas of bones cannot be made. These lesions present the same histologic characteristics as fibrous dysplasia. They are considered by many authors[8,15,16,18,19,25] as the same disease and not separate entities.

It becomes readily apparent that the pathologic diagnosis can be confusing. It would seem that fibrous dysplasia has microscopic variations which some authors have designated by different terminology. Actually, they all appear to be the same pathologic process.

The pathologic slides and material to confirm the diagnosis in cases used in this report were reviewed by a number of pathologists, and the diagnosis of fibrous dysplasia was made without hesitation.

PATHOGENESIS

In reviewing various articles on fibrous dysplasia, with particular reference to origin and development of the disease, one can soon find diversified viewpoints. The various investigators of the pathogenesis have opposing concepts, all in their own right correct, but all basically different. In considering the more important and more frequently discussed explanations of the pathogenesis, one will immediately be impressed with the difficulty and confusion that exists in defining the limits of this disease process. As previously stated, there exist three forms of fibrous dysplasia, the monostotic form, the polyostotic form, and the endocrine form (Albright's disease). The assumption that all three of these processes have the same etiology and one is just a variation of the other is not generally accepted. There are also viewpoints that state the endocrine form of fibrous dysplasia is

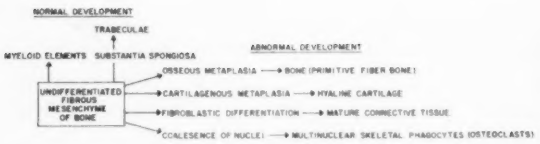
NORMAL DEVELOPMENT
TRABECULAE
MYELOID ELEMENTS
SUBSTANTIA SPONGIOSA
ABNORMAL DEVELOPMENT
OSSEOUS METAPLASIA → BONE (PRIMITIVE FIBER BONE)
UNDIFFERENTIATED FIBROUS MESENCHYME OF BONE
CARTILAGENOUS METAPLASIA → HYALINE CARTILAGE
FIBROBLASTIC DIFFERENTIATION → MATURE CORRECTIVE TISSUE
COALESCENCE OF NUCLEI → MULTINUCLEAR SKELETAL PHAGOCYTES (OSTEOCLASTS)

FIG. 1. Diagram showing the multipotent action of the undifferentiated fibrous mesenchyme of bone.

unrelated to the single bony lesions.[25] This diversified thinking can and does lead to a dilemma. It is best to consider the more commonly discussed concepts. In all of these premises, there are gaps in continuity which cannot be filled with proved scientific fact. At times, in a particular patient, each of these theories may be logical and correct, but no one viewpoint seems to cover all cases. There is no evidence to support the assumption that the lesions in the skull or facial bones have a different pathogenesis than in bones elsewhere in the skeleton.

CONGENITAL RELATIONSHIP

Lichtenstein,[13] in 1938, described 4 cases, all of which were documented with biopsy and complete clinical investigation. He proposed the term, "polyostotic fibrous dysplasia." His original description of the pathogenesis is still classical. It has been usurped by none. The basis of his rationale is, " . . . a disturbed function or development of the bone-forming mesenchyme,

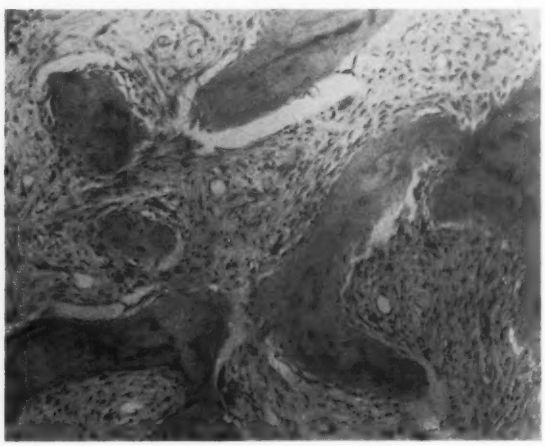

FIG. 2. Photomicrograph of fibrous dysplasia. The lesion is composed of dense fibrous tissue and irregular immature bone spicules.

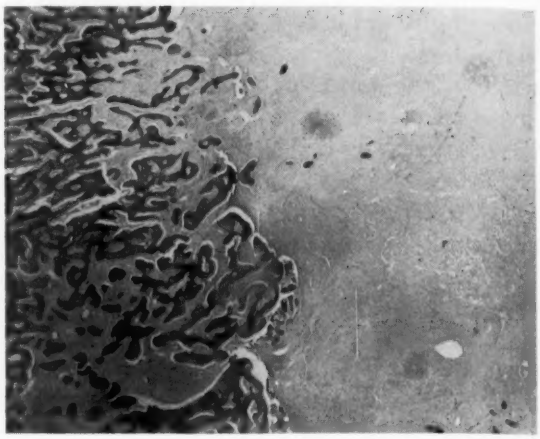

FIG. 3. Photomicrograph of fibrous dysplasia. The left side reveals dense trabeculae and fibrous tissue. The right side is composed of dense fibrous matrix and only an occasional small bone spicule. This biopsy material readily explains the dense bony areas and the radiolucent fibrous areas seen roentgenographically.

which results in replacement of the substantia spongiosa* and filling of the medullary cavity of affected bones by fibrous tissue in which trabeculae of poorly calcified primitive new bone are developed by osseous metaplasia." It is further stated that a clearer understanding can be had if one realized the multipotent action of the undifferentiated fibrous tissue, which under normal development forms the substantia spongiosa and myeloid elements of bone, but under abnormal circumstances, may lead to the following pathologic transformations:

1. The undifferentiated mesenchyme by direct action may form osteoid or primitive fiber bone. This process is called metaplasia, and since bone is the formed substance, it is termed, "osseous metaplasia."
2. Cartilaginous structures may also result from metaplasia.
3. The fibrous mesenchyme may become more differentiated and produce mature collagen tissue.
4. Multinucleated cells, which are thought to represent osteoclasts, may

* The spongy substance of bones composed of crossing trabeculae.

form by the coalescence of nuclei of the fibrous mesenchyme.

Figure 1 diagrammatically represents this process.

Lichtenstein continues by stating that in the involved areas which are poor in vascularity, the fibrous tissue presents more collagen and less bone; conversely, the well vascularized areas apparently have more bone fiber. In addition, the cases presenting the most extensive osseous involvement will have the basic fibrous tissue show a more pronounced osteogenic differentiation. The etiologic agent directly responsible for the osseous malformations is not known; however, since the disease begins in early life and has a tendency to involve one half of the skeleton and skull, the suggestion of the congenital factors deserves consideration.

Falconer, Cope and Robb Smith[7] have advanced a proposal that the precocious puberty seen in the endocrine form of fibrous dysplasia is similar to the type seen in congenital lesions of the hypothalamus, such as hamartomas and craniopharyngiomatous cysts. They state that these lesions are visible macroscopically and that there is no apparent reason why a smaller congenital lesion in the hypothalamus should not produce the same endocrine effect. Therefore, the congenital basis of the osseous manifestations and the congenital hypothalamic lesions can be a method of explaining this syndrome, and this " . . . is most easily regarded as an error or early embryological development."[7]

RELATIONSHIP TO TRAUMA

Schlumberger[25] has advanced a plausible explanation of pathologic changes which occur in the monostotic form, all of which can be observed microscopically. He feels, as most observers do, that the primary component of these bone lesions is connective tissue, and the pathologic reaction is metaplasia; that is, the formation of bone directly from fibrous tissue. The chronologic order of events is as follows: the connective tissue cells which are fibroblasts change from an elongated spindled appearance to become rounded. The fibrils in the intercellular matrix thicken and readily stain with eosin. Thereafter, the intercellular substance becomes more abundant and incarcerates connective tissue cells which resemble osteocytes. The tissue then can be identified as osteoid. The connective tissue bundles change from fibrous tissue to the mature bone trabeculae, revealing the fibrous metaplastic origin of bone. Concomitantly, other observations in the pathogenesis are noted. The connective tissue cells, after becoming more spherical, may be located at the periphery of the metaplastic bone and act as osteoblasts. Osteoclasts, which can be large and multinucleated, will also be observed breaking down the newly formed bone. This local destruction can lead to the formation of small cysts.

Schlumberger also points out that while metaplasia is the main feature of fibrous dysplasia, all of the connective tissue in these bone lesions does not have this property. He continues by saying there is no histologic difference in the connective tissue which has a tendency to form bone as compared to the connective tissue that does not have this ability. The factor of trauma in monostotic fibrous dysplasia should be considered as a possible etiologic agent, since one-third of the cases presented this history. It should be noted, however, that there is no proof that the lesion did not exist prior to the injury. In all of the cases examined, islets of cartilage were not found except where there was evidence of pathologic fracture. This last observation has not found unanimous agreement since small islets of cartilage within the lesions of fibrous dysplasia have been seen by many observers.[2,13,23,24] Their hypothesis is that osseous tissue and cartilage tissue may be formed by fibrous metaplasia and may follow a somewhat similar gross anatomic pattern. We, therefore, find a relationship between fibrous dysplasia and multiple skeletal enchondromata (Ollier's disease). Schlumberger points out that the 67 cases he analyzed were cases that had

single bone involvement. None were of the polyostotic variety, and none presented hyper-pigmentation of skin. He concluded that his cases did not support the belief that polyostotic and monostotic fibrous dysplasia are different stages of the same disease. He gains support from Bremer[3] and Scholder[26] and states the polyostotic lesions seen in young girls affected with precocious puberty may be related to a high blood estrogenic titer acting through the parathyroids. Two of his cases were eliminated from the study because of polyostotic involvement; 1 was a female. Of the 67 reported monostotic cases, 4 were females. It would seem logical to assume that in any series where 64 of the cases are male, there would be no severe forms of the disease with endocrine dysfunction since this form of the disease (Albright's disease) is almost exclusively limited to females. To propose, therefore, that Albright's disease is the probable origin would seem illogical when 94 per cent of the patients studied were males with single bone lesions. Warrick[29] has presented 2 examples of fibrous dysplasia in males with precocious sexual development.

Direct trauma prior to the onset of the disease was observed in this series four times and seems to be of questionable significance. It may not always be a factor in initiating the disease from a clinical viewpoint, but in 1 case was related to subsequent acceleration of growth of an already existing fibrous dysplasia. A strong argument against trauma as an etiologic factor is that the microscopic sections consistently fail to show evidence of recent or old hemorrhage.

RELATIONSHIP TO NEUROFIBROMATOSIS OF VON RECKLINGHAUSEN

In 1944, Thannhauser[28] presented a detailed report showing that neurofibromatosis and osteitis fibrosa cystica have a common pathogenesis. His teachings, apparently well documented and thorough and carrying a connotation of European investigators, have not been generally accepted by American writers. As I will subsequently point out, Jaffe, an American author, replied to Thannhauser's article and specifically stated in his title that fibrous dysplasia of bone is not an expression of neurofibromatosis. Jaffe[11] states that all cases of neurofibromatosis do not affect the skeletal system, and the osseous involvement by neurofibromatosis is not uniform and does not follow a constant pattern. Furthermore, skeletal abnormalities which are seen in neurofibromatosis such as congenital anomalies are not, as a rule, associated with fibrous dysplasia. The more common ones may be listed as follows: kyphoscoliosis, spina bifida, congenital dislocation of the hip, absence of extremity bones, etc. The main premise that causes contention about the osseous pathogenesis is that Thannhauser states, " . . . nerve elements are not present in the histological specimens of fibromatous bone lesions in both conditions, yet whorls of spindle cells are found in the bone fibroma in neurofibromatosis as well as in some areas of the fibrous tissue in osteitis fibrosa cystica localistata and disseminata. These whorls of cells are also present in cutaneous neurofibromata."

This is indicative of a neurofibromatosis origin because in the center of such whorls, a nerve fiber with schwannoglia was once present and initiated the fibrous growth. The presence of osteitis fibrosa cystica localisata and disseminata in some fibrous tissue areas gives additional histologic confirmation to the theory that the pathogenesis of neurofibromatosis (von Recklinghausen) and that of osteitis fibrosa cystica localisata and disseminata are related. Jaffe replied to this theorizing with strong and pointed statements. First, the whorling appearance of connective tissue is nonspecific, and does not indicate its neurogenic origin. Next, the basic fibrous tissue of these bone lesions can exhibit the ability to undergo metaplasia to osseous tissue and a less extent to cartilage. This metaplasia has never been demonstrated in lesions of neurofibromatosis of the skin, subcutaneous tissue, nerve structures or within bone. Further, Jaffe states that neurofibromatosis lesions

have a characteristic histologic appearance based on degenerating nerve fibers and, if they are few, on the microscopic appearance of formations of the nature of schwannian hypertrophies. The lesions of fibrous dysplasia do not demonstrate degenerating nerve fibers or other characteristic features of neurofibromatosis such as the palisading of nuclei.

RELATIONSHIP TO LIPOID GRANULOMATOSIS

In this textbook, Snapper[27] proposes an explanation for the pathogenesis of fibrous dysplasia, referring for the most part to the polyostotic form. He presents 3 cases from which he assumes that fibrous dysplasia is a result of a ". . . burnt-out case of lipoid granulomatosis of bone." His first case appears to represent the typical features of polyostotic fibrous dysplasia with endocrine dysfunction, as observed by Albright. There were no clinical features of Hand-Schüller-Christian disease (lipoid granulomatosis), except on one occasion a hypercholesteremia was noted. A biopsy of an involved femur is said to have revealed the microscopic appearance of fibrous dysplasia; however, there were foam cells that stained positive for fat and lipoid particles outside of the foam cells in the fibrous tissue. A photomicrograph of this, his most convincing case, was not published. Therefore, without clinical evidence of lipoid granulomatosis, plus only one positive nonspecific laboratory examination and an unpublished photomicrograph of the biopsy, we cannot be certain that this case was Hand-Schüller-Christian disease.

His second case was also clinically typical of polyostotic fibrous dysplasia. Two biopsies verified this diagnosis. There was slight elevation of the serum cholesterol; and, therefore, a third biopsy was decided upon. The previous biopsies were on old lesions in which fractures had occurred, thereby probably obscuring the underlying pathology. The third biopsy was performed in a more recent lesion. Again, the microscopic lesion of fibrous dysplasia was demonstrated; however, in addition, xanthoma cells with a foam structure were seen. Sudan III stain revealed crystals; therefore, the diagnosis of lipoid granuloma had to be considered.

In the third case, Snapper again presents a typical case of polyostotic fibrous dysplasia, this time in a thirty-one year old man. The serum cholesterol was elevated to 380 mg., 420 mg., and 480 mg. The roentgenograms demonstrated findings diagnostic of fibrous dysplasia, and a skull biopsy confirmed these findings. The biopsy of a small bone lesion in a proximal phalanx revealed the presence of lipoid granuloma. There are only two features in these cases that even vaguely justify a consideration of lipoid granulomatosis: one, an occasional elevated blood cholesterol; and two, the presence of foam cells containing fat, occasionally observed in biopsies. Point one is of minimal significance, not diagnostic and overstressed. Point two is also not important since occasional foam cells are seen in many bone diseases, including fibrous dysplasia.[25] When they occur in small numbers, they are not diagnostic any more than a few giant cells are diagnostic of a giant cell tumor.

Furthermore, the skull lesions of lipoid granulomatosis have a roentgen pattern different from fibrous dysplasia. In only one case in this entire series were the roentgen findings in the skull multiple, rounded, and nonexpansile defects (Fig. 4). The remaining cases all showed expanding bone producing lesions. The biopsy and clinical course of the patient represented in Figure 4 were in no way suggestive of Hand-Schüller-Christian disease. This patient was nine years of age and demonstrated precocious sexual development with the onset of menstruation at four and a half years. The blood cholesterol was 161 mg. per cent, and there was no evidence of exophthalmos. The peripheral bones, with lesions predominantly on the right side were typical of fibrous dysplasia. The biopsy did not confirm lipoid granulomatosis.

FIG. 4. This nine year old white female presents the typical findings of Albright's disease. Menstruation began at $4\frac{1}{2}$ years of age and occurred three times till the age of nine. She suffered numerous pathologic fractures of the extremities. On physical examination there were no café au lait spots; however, there was precocious sexual development. The blood chemical studies were within normal limits including a blood cholesterol which was 161 mg. per cent. The roentgen findings in the skull, numerous round destructive areas, are somewhat suggestive of a lipoid granulomatosis; however, biopsy of the peripheral bone revealed findings that were characteristic of fibrous dysplasia.

ROENTGEN FEATURES

The roentgen findings of fibrous dysplasia of the skull and facial bones are of unquestionable significance. It is true that the microscopic diagnosis cannot be made on a roentgenogram, but the roentgenogram does clearly define the limits and characteristics of the gross pathology. Many biopsies are small and contain only fragments of tissue; consequently, the gross characteristics of the resected tissue are limited. Many of the gross features of this disease, which can be studied only roentgenographically, impress us with a characteristic pattern that at times appears diagnostic.

In reviewing all of the cases, it was found that 18 involved only the skull, 5 involved both the skull and facial bones, and 16 involved the facial bones alone. Gray's Anatomy[10] includes the following as skull bones: frontal, parietal, temporal, sphenoid, occipital, and ethmoid. The facial bones are listed as: nasal, maxillary, lacrimal, zygomatic, palatine, concha, and vomer. Gray also mentions that the frontal, occipital, and vomer are unpaired. Table 1 shows the frequency of involvement of these bones. Four patients or about 10 per cent presented skull or facial bone involvement that was not predominantly unilateral. One of these patients had extensive lesions that involved all of the cranial or facial bones. The remaining 35 patients or 90 per cent had lesions that were predominantly unilaterial. This point is of doubtful significance because a small lesion has a much greater chance to be on one side than in the midline. In reviewing all of the cases, it became evident, that the lesions of the skull and facial bones could be divided into three categories by their roentgen appearance alone. These types are: (1) pagetoid, (2) sclerotic, and (3) cyst-like. In an occasional instance, the division of roentgen findings into three forms may be difficult, since a lesion may present some criteria of each type. These terms are merely descriptive and are various roentgen manifestations of the same disease.

The first type, called the pagetoid type, is so named because of its roentgen similarity to Paget's disease. The involved area is expanded and has alternate areas of density

TABLE I

Bone	No. of Times Involved
Occipital	10
Parietal	20
Frontal	29
Temporal	16
Sphenoid	25
Ethmoid	25
Nasal	15
Maxillary	16
Lacrimal	18
Zygomatic	8
Palatine	7
Concha	6
Vomer	6

Note: The occipital, frontal and the vomer are unpaired and therefore are tabulated once. All other bones occur in pairs and are thus tabulated twice if both sides are involved in each patient.

and radiolucency. The skull may obtain a thickness of more than seven centimeters. Most of the expansion is outward with thinning, erosion, and disappearance of the outer table. These have been mentioned as valuable signs by other authors. Osteoporosis circumscripta does not occur. Figures 5, *A* and *B* and 6 are typical roentgenograms of patients with this type of involvement. Occasionally, a skull lesion is best described as a "blister-like" lesion (Fig. 7).[30] This type of close inspection

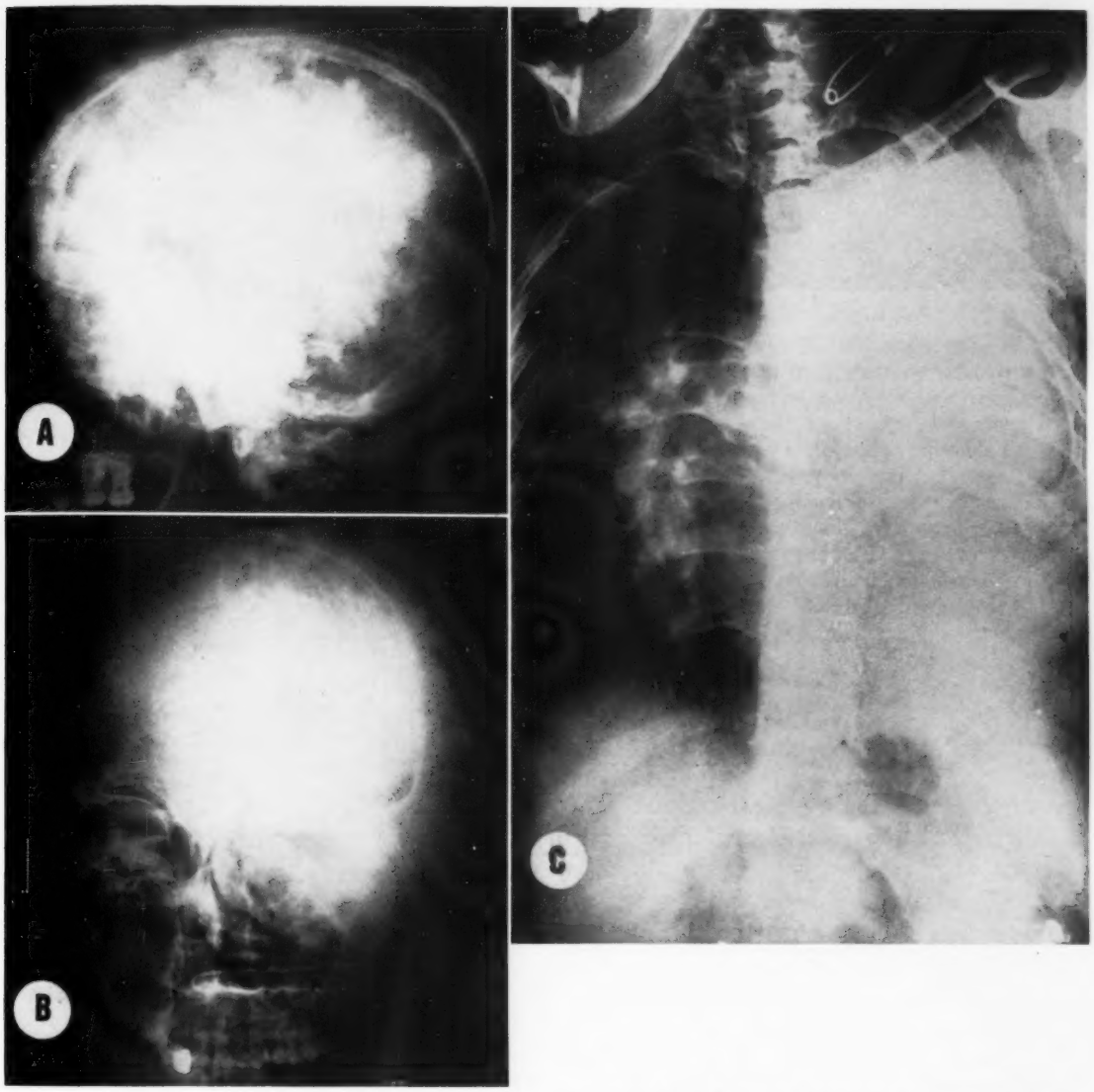

Fig. 5. This thirty-one year old colored female presents the typical findings of Albright's disease with fibrous dysplasia of bone, and precocious onset of puberty with the menarche at the age of one year. A definite asymmetry of the skull and face have been noted for many years (as long as the patient can remember) which is limited to the left side. There was also proptosis of the left eye. A large mass involved the left hemithorax and deviated the trachea and heart to the right. The roentgenograms of the skull (*A* and *B*) show a tremendous thickening of the cranium which in the widest area measures approximately 7 cm. The chest lesion (*C*) remained the same over a four year observation period. Exploratory thoracotomy revealed a hard mass obliterating the entirety of the left thoracic cage. Biopsy of the mass revealed fibrous dysplasia. This large mass in the thoracic cavity originated from the upper ribs on the left side and grew into the thoracic cage producing the deformity.

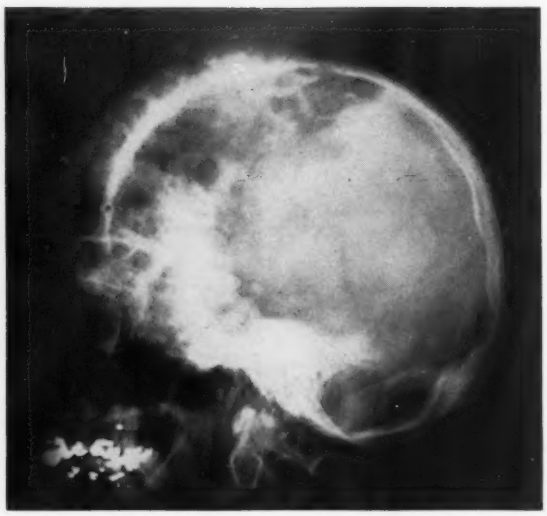

FIG. 6. This fifty-one year old white female has complained of headaches for three years. Trauma was said to have occurred to her head at the age of fourteen with resulting depression of the left orbit. Previous examinations have substantiated a schizophrenic behavior. The blood chemical studies were within normal limits. The roentgenograms revealed the finding mostly limited to the left side of the face and skull with definite thickening of the involved bone and apparent encroachment on the inner table. This type of lesion resembles Paget's disease and is appropriately called the pagetoid type. The remaining osseous system failed to reveal evidence of peripheral lesions.

has alternate areas of density and radiolucency and may be suggestive of a "cotton wool" appearance. This type of lesion presents more features of the pagetoid type and therefore is classified as such. Twenty-two patients or 56 per cent demonstrated the pagetoid form. It is noteworthy to mention that the inner table involvement may be more marked than the outer table involvement. This fact has not before been stressed in the literature. Figure 8 presents a lesion involving the horizontal table of the frontal bone. It projects about two and a half centimeters into the cranial cavity and less than one centimeter toward the roof of the orbit.

The second type of roentgenographic lesion is the sclerotic form. These lesions present a homogeneously dense area of involvement. Expansion of the bone is also noted with the outer table being most often involved, but again the inner table may be affected to the same degree as the outer table. Figures 9 and 10 illustrate examples of the sclerotic form. Nine patients or 23 per cent demonstrated the sclerotic form.

The cyst-like lesions comprise the third category. These are round or oval in shape and usually just one lesion is present. They are most frequently seen in the skull; only one case involved the facial bones. These lesions commonly have a dense border that

FIG. 7. A thirty-six year white female complained of an enlargement of the left side of her head for about six months' duration. Sexual development was not precocious and the patient thought menarche was at the age of twelve. The roentgenograms demonstrated a widening of the parietal bone on the left side with thinning of the outer table. This lesion was biopsied and found to represent fibrous dysplasia. The remaining portion of the skeleton was within normal limits and the blood chemical studies were not unusual. This type of lesion is characteristic of fibrous dysplasia when occurring in a young person. It represents the pagetoid type, because of the "cotton wool" appearance and gross similarity to Paget's disease. This has been described by some as having a "blisterlike" appearance.

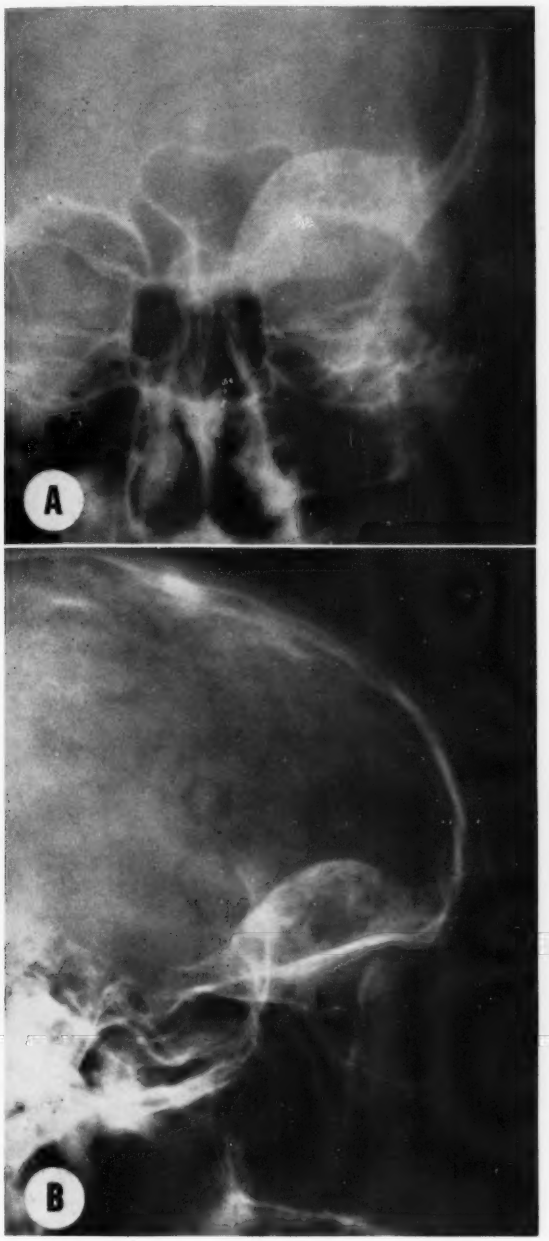

FIG. 8. A white male of twenty-four years was asymptomatic except for slight swelling over the left eye. Routine skull examinations (*A* and *B*) revealed a lesion in the region of the horizontal portion of the frontal bone with definite intracranial extension. There is involvement of the left sphenoid bone in the region of the anterior clinoids. This lesion has alternate areas of density and radiolucency and only slightly expands the outer portion of the skull in the region of the roof of the orbit. This was biopsied and proved to be fibrous dysplasia.

is thin and distinct. Their average size is between two and five centimeters and both tables of the skull may be involved. Figure 11 illustrates a patient with a single lesion. It is interesting to point out that this was the only lesion this patient possessed. It was discovered on a complete bone survey during the follow-up of an established diagnosis of carcinoma of the thyroid. Biopsy proved the lesion to be fibrous dysplasia. On initial inspection, one may say this is an example of the monostotic form of fibrous dysplasia; however, this small, single lesion unquestionably involves the frontal and parietal bones, thereby making it technically polyostotic. Apparently, the sutures in the skull and facial bones do little to restrict or limit the growth or expansion of a lesion of fibrous dysplasia. This observation is in contrast to peripheral bone lesions where the epiphysis and joints are seldom primarily involved by fibrous dysplasia.

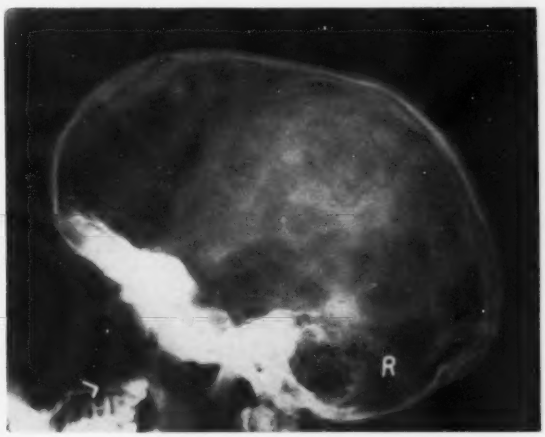

FIG. 9. A thirteen year old white female complained of headaches during the past five years. There has been slight proptosis of the left eye during this time and prominence in the left orbital regions. For the past one year, there has been progressive loss of visual acuity to almost complete blindness. On physical examination, there was a fullness of the left orbital region and reduced vision in the left eye to light perception. No other defects or abnormalities were noted. The roentgenograms demonstrated a diffuse homogeneous enlargement of the base of the skull (most marked on the left side). The optic foramen on the left was definitely diminished. The treatment consisted of decompression of the left optic nerve.

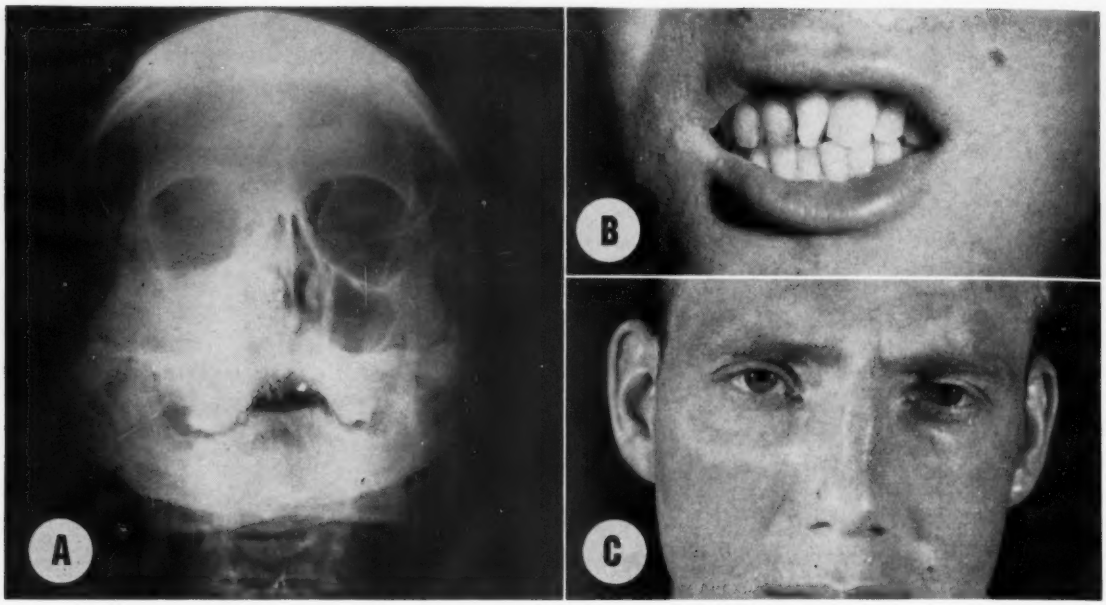

Fig. 10. This twenty-one year old white male complained of a diffuse, non-tender enlargement of the entire right maxillary region. He also stated that the right central incisor tooth had slowly become rotated. These symptoms had been present for about three months. The roentgenogram (*A*) demonstrates involvement of the right frontal and ethmoid bones. The right maxilla is involved with widening of the bone and obliteration of the maxillary sinus. The involvement also extended to the alveolar portion of the right maxilla. The photograph (*B*) demonstrates the rotation of the incisor tooth. The photograph (*C*) illustrates a definite fullness on the right face due to the expanding maxillary fibrous dysplasia. The roentgen findings are characteristic of fibrous dysplasia. The biopsy confirmed the diagnosis.

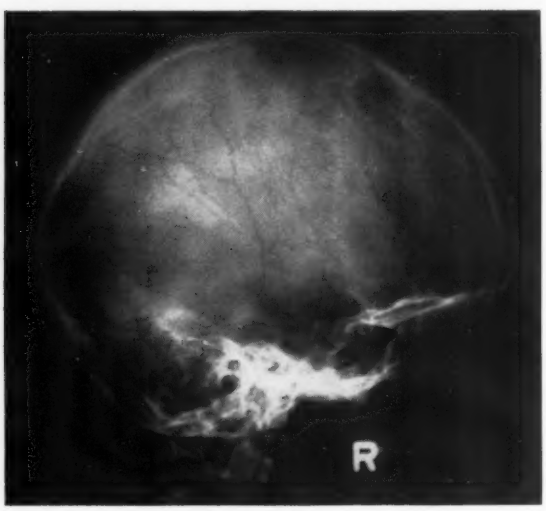

Fig. 11. This twenty-seven year old white female has had numerous neck operations since the age of seven because of an adenocarcinoma, Grade 1, of the thyroid gland. On a bone survey a rounded defect was noted in the calvarium which was thought to represent a bony metastasis. The lesion, being the only bone lesion present, was resected and found to represent fibrous dysplasia. It measured approximately 2 cm. in diameter and involved both the frontal and parietal bones, therefore representing a polyostotic lesion.

Figure 4, previously discussed in the relationship to lipoid granulomatosis, is another example of the cyst-like variety. Cyst-like lesions were not found in the base of the skull. Eight patients or 21 per cent demonstrated the cyst-like lesions.

Two cases presented involvement of the frontal bone with limitation to the region of the frontal sinus. The roentgen appearance is that of an extensive frontal sinusitis with dissolution of the mucroperiosteum and adjacent frontal bone osteitis. The biopsies revealed the typical fibrous dysplasia, and no evidence of an inflammatory reaction existed. Both of these patients had enlargement of the forehead over the involved region. Figure 12 is an example of fibrous dysplasia, the limitations of which are in the region of the frontal sinus.

Fig. 12. A twenty-eight year old white male for the past three years noted an enlarging mass in the region of the left eyebrow and glabella. The patient also complained of generalized headaches. The roentgenograms revealed prominence in the region of the frontal bone and increase in density overlying the region of the frontal sinus. This is suggestive of an osteitis of the frontal bone subsequent to a frontal sinusitis. This mass was biopsied and found to represent fibrous dysplasia. The headaches, probably unrelated to the osseous involvement, were due to an arteriovenous aneurysm in the posterior cerebrum which was demonstrated by angiography.

Table II

ROENTGENOLOGIC TYPE OF BONY LESION

	Pagetoid	Sclerotic	Cyst-like
Per cent of total cases	56	23	21
Average number of skull and/or facial bones involved per patient	6.4	3.6	2.7
Average age of patients in years	32.9	18.2	22.6
Total duration of symptoms in years	14.8	2.7	0.5

DISCUSSION

In studying these 39 patients from a clinical, roentgenologic and pathologic point of view, we find interesting observations when comparing the features of the three types of roentgen lesions; Table II summarizes these data. We notice the pagetoid type was found in more than one half the total number of patients studied (56 per cent). The other two types of lesions, the sclerotic and the cyst-like, occurred about the same frequency, 23 per cent and 21 per cent, respectively. The pagetoid type presents an average of 6.4 skull and/or facial bones involved per patient. This indicates the pagetoid form is more extensive since about two to three times as many bones are involved.

Next, the average age of the patients in these categories is noteworthy. The individuals with the pagetoid type have an average age of 32.9 years. The age used in this determination was the age when the patient underwent the clinical investigation that is available for the author's review. The other two forms contained patients of a much younger average age group. The sclerotic type averaged 18.2 years and the cyst-like 22.6 years. The pagetoid patient on the average is ten to fourteen years older than the other two groups. Along this same channel of thought, we find the total elapsed time from the first sign or symptom until the patients presented themselves for investigation was 14.8 years for the pagetoid type, 2.7 years for the sclerotic type, and 0.5 years for the cyst-like type. From these figures, we notice that in the skull and facial bones, the pagetoid type of fibrous dysplasia is the most common, and since it involves more bones than the other types it is the most extensive. It occurs in an older individual, as a rule, and the duration of symptoms is definitely longer.

We have little information on the long term follow-up for roentgen comparison. This would aid materially in observing the course of the natural history of this disease. It would seem possible that the sclerotic and cystic types are the early phase or the forerunner of the pagetoid type. This is demonstrated in one case, followed for a period of thirteen years, initially showing the sclerotic form of the disease followed by a slow transformation to the pagetoid type.

Although this point is only conjecture, it is certainly feasible, since all of these patients have the same disease—fibrous dysplasia.

The most common symptom was a local swelling or asymmetry of the face or head. This was present in 31 patients or 79 per cent. Every one of these 31 patients had symptoms over the areas of bony involvement.

There were 12 cases that presented proptosis or exophthalmos. These same cases had a definite asymmetry of their face. The right side was involved 5 times and the left side 7 times. In all 12 cases, the frontal bone was involved; the sphenoid was involved in 11 patients; the ethmoid was involved in 9 patients; and the lacrimal bone in 7 patients. Involvement of the maxilla *per se* in this series did not cause deviation of the eye. Most of the patients (8 of the 12) had the pagetoid type, 3 had the sclerotic type, and 1 case had the cyst-like type.

Six patients presented symptoms referable to the nose. All 6 patients complained of nasal obstruction, in 2 cases bilateral and in 4 cases, unilateral. In 1 of these cases, there was a unilateral bloody nasal discharge. Five of the 6 patients had asymmetry of the face. In all 6 cases, the ethmoid bones were involved. In 5 cases, the lacrimal bone was involved. The nasal and maxillary bones were affected in 4 patients. The concha was invaded 3 times, and the vomer and palatine were involved in 2 patients, respectively. Figure 13 is a roentgenogram of a patient that complained of unilateral nasal obstruction and a bloody nasal discharge of three weeks, duration. The homogeneous density involving the left nasal cavity was due to a mass involving only the left middle turbinate. This measured $1 \times 1.5 \times 2$ cm. in size. It was completely excised and the microscopic diagnosis was fibrous dysplasia. Six years later, the patient was asymptomatic and without recurrence or additional lesions.

Pain was a difficult symptom to evaluate. It occurred in 20 patients (51 per cent) and was believed to be related to the lesions of fibrous dysplasia. In view of the difficulty in separating pain from tenderness and from headaches, a more elaborate analysis of this symptom is not warranted.

FIG. 13. This twenty-two year old white male complained of left nasal congestion and repeated nasal bleeding of three weeks' duration. On examination, a large pink mass was noted obstructing the nasal cavity. At operation, this mass was found involving the entirety of the middle turbinate. It was resected and there was no recurrence six years later. The roentgenogram revealed an obstruction of the left nasal cavity which appeared to be homogeneous. No other lesions were found on the bone survey. The microscopic diagnosis was that of fibrous dysplasia which was limited to the middle turbinate.

In this series, 20 patients were males, and all were white. Nineteen patients were females of which 17 were white and 2 were colored.

The 2 cases with extensive involvement of the frontal bone were also schizophrenic. In each case, the bony involvement preceded the abnormal behavior by many years. In both of these cases, the roentgenograms were almost identical (Fig. 6), and they presented the following features: (1) both had the pagetoid type; (2) the involved area was the vertical and horizontal part of the frontal bone (floor of the anterior cerebral fossa) and the sphenoid bone (floor of the middle cerebral

fossa); (3) the inner table was deformed, and the cranial contents were encroached upon. Both patients were women, and no bony lesions outside of the skull were found. Each of these cases also exhibited asymmetry of the face with proptosis of one eye. Their menstrual history was normal. It is not possible to state if the bony lesions were related to the psychotic behavior of these patients. A comparison can be made, however, to the gross bony involvement, which is similar in every respect to Paget's disease. Mental disturbances, such as senile psychosis and convulsions associated with Paget's disease of the skull, have been previously described.[9,17] These gross changes in Paget's disease reveal widening of the skull and compression of the brain. Goldenberg's[9] publication illustrates a coronal section of the skull and brain of a patient with Paget's disease. The widening of the skull, especially in the frontal region resulted in encroachment of the frontal lobes with resultant atrophy. The gross pathology is, in every way, similar to the 2 patients with fibrous dysplasia. It is known that central nervous system changes are associated with fibrous dysplasia. These are mainly limited to children and appear to be problems of mental deficiency, rather than a psychosis starting during adult life.[12] Since the clinical findings and roentgenograms in both cases are so strikingly similar, I feel this point deserves to be mentioned.

Peripheral regions, other than the skull or facial bones, were involved in 6 patients or 15 per cent. In all of these cases, the involvement of the cranium was more extensive than average with 11.5 skull or facial bones affected. Five of the 6 patients demonstrated the pagetoid form, and 1 case, multiple cyst-like areas in the cranium. Four of these patients presented Albright's disease, and they were the only manifestations of Albright's disease in the entire series. In this study, 11 cases or 28 per cent presented one bone involved (monostotic), and 28 cases or 72 per cent presented more than one bone involved (polyostotic). It must be remembered that in the 28 cases presenting polyostotic forms, 6 cases had peripheral involvement, in addition to the multiple skull or facial bones involved.

The alternate areas of density and radiolucency in the pagetoid type can be explained by realizing the dense areas are composed of more compact, mature, calcified, and trabeculated bone than the radiolucent areas which are richer in fibrous tissue with little or no bone or poorly formed osteoid. Figure 3 is a photomicrograph that gives support to these statements. One side of the section shows bony trabeculae which are dense and close together. A sharp line of demarcation exists, and the other half of the lesion is composed of fibrous tissue with only an occasional bony trabecula. This biopsy material readily explains the dense bony areas and the radiolucent fibrous areas seen roentgenographically. The sclerotic cases present lesions in which the trabeculae are mature, and less fibrous tissue is present. Although the microscopic field may demonstrate many small irregular bone trabeculae, their gross over-all arrangement is uniform and the amount of calcium deposited in the trabeculae is apparently high (Fig. 2). This will therefore result in a roentgenogram in which the lesion is homogeneously dense and aptly called the sclerotic type (Fig. 10).

The cyst-like lesions presented bony trabeculae throughout their specimens; however, the trabeculae were not as dense per unit area. As a rule, they demonstrated a more immature type of bone; *i.e.*, osteoid. This type of lesion will be more radiable than the normal adjacent bone. The peripheral lesions have a closer resemblance to the cyst-like type than to the other forms.

The therapy of these lesions, if possible and practicable, is complete excision. This was performed on 6 patients with single lesions, and 2 of the patients were followed at least two and six years, respectively. They show no recurrences or new lesions. All other forms of surgery, while not cura-

tive, are important. The patient in Figure 9 underwent surgery, and the roof of the orbit was removed so that the optic nerve at the optic foramen could be decompressed. Patients who appear to do well without any treatment are those with large lesions that do not involve vital structures and are not progressing in size. The patient presented in Figure 5 remained the same clinically and roentgenographically during her entire time of observation of four years. Roentgen therapy was given to 1 patient with maxillary bone involvement because of pain. A total of 400 r was given—100 r per treatment for four treatments. The factors were 200 kv., half-value layer 1.5 Cu. There was relief from pain for six months; however, the bony involvement slowly progressed. One case has been reported[6] where a favorable response to the relief of pain was obtained with cortisone.

The additional disease processes presented by these patients should be mentioned. One patient, a white female, had adenocarcinoma of the breast that developed at the age of thirty-five. She had polyostotic involvement, and during adolescence, she suffered numerous pathologic fractures of the femur. Another patient, a white female previously mentioned, presented an adenocarcinoma of the thyroid. This diagnosis was established at the age of seven years, and at the age of twenty-seven, a small asymptomatic calvarial defect was noted on a bone survey (Fig. 11). The patient illustrated in Figure 5, *A* and *B*, a colored female of thirty-one years with a typical example of Albright's disease, presented a large dense mass filling the entire left thoracic cage. Figure 5*C* is a chest roentgenogram of this patient. The trachea and cardiac shadow are shifted to the right. No change was noted in this lesion over a four-year period. An exploratory thoracotomy revealed a large, hard, bony mass filling the entire left upper thoracic cage. It was difficult to separate this mass from the chest wall. The biopsy in multiple areas presented the typical microscopic appearance of fibrous dysplasia. It apparently arose from the markedly deformed third posterior left rib, since no other bony abnormalities existed in the left thoracic cage or in the thoracic spine. A large lesion such as this has not been reported previously. One patient, a twenty-eight year old male (Fig. 12), had an arteriovenous aneurysm of the occipital lobes which was proved by angiography and craniotomy. Diabetes mellitus was observed in a white male, aged thirty-nine.

Complete blood chemistry studies were not performed on all patients; therefore, a detailed evaluation is not practicable. Occasionally, as other observers have noted,[22] the alkaline phosphatase was elevated in the patients having the most extensive form of the disease. The blood calcium and phosphorus were normal.

SUMMARY

1. Thirty-nine cases of fibrous dysplasia of the skull or facial bones, all with microscopic confirmation, have been subjected to a critical analysis.

2. The pathology has been reviewed. The basic tissue disturbance exists in bone, whereby fibrous tissue has the ability to form bone directly (metaplasia).

3. When fibrous dysplasia involves the skull or facial bones, the roentgen examination shows three forms to be distinguishable. The classification suggested is: (1) pagetoid, (2) sclerotic, and (3) cyst-like.

4. The pagetoid type, in contrast to the other two types, is the most common, has the most extensive involvement in reference to the number of bones, occurs in a slightly older age group, and presents symptoms of longer duration.

5. Eleven cases (28 per cent) presented one skull or facial bone involved (monostotic), and 28 cases (72 per cent) presented more than one skull or facial bone involved (polyostotic).

6. The most common symptom was swelling or asymmetry of the head or face,

occurring in 31 or 79 per cent of the patients.

7. Twelve cases presented unilateral proptosis. In all of these cases the frontal bone was involved, and in 11 cases the sphenoid bone was involved.

8. Six patients presented nasal symptoms, the most constant being nasal obstruction. In all of these cases the ethmoid bone was involved, and in 5 cases the lacrimal bone was involved.

9. Pain was difficult to evaluate, but occurred in about one-half of the cases.

10. Severe mental disturbances were observed in 2 patients with extensive frontal bone changes. The bony encroachment on the frontal lobes with resultant atrophy is a logical explanation.**

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MULTIPLE DIAPHYSIAL SCLEROSIS*

REPORT OF A CASE OF POSSIBLE TYPHOID ETIOLOGY

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BONE lesions due to typhoid fever have been well documented by many authors.[2] One of the rarer types of bone typhoid, multiple diaphysial sclerosis, has been the subject of occasional reports.[1,6]

In recent years there have been several reports[4,5] of groups of cases of multiple diaphysial sclerosis of unknown etiology. In this communication a case is presented showing bone changes apparently secondary to typhoid fever complicated by draining sinuses in the lower extremities. The relations between this condition and other reported instances of multiple diaphysial sclerosis are discussed.

REPORT OF A CASE

L. S., a Chinese female nurse, aged twenty-eight, was admitted to Michael Reese Hospital on February 9, 1953 for a medical evaluation because of slow resolution of a left sided pneumonia and the development of an empyema pocket in the posterior axillary region. Studies for tuberculosis were completely negative. On follow-up in the out-patient chest clinic, slow progressive clearing of the empyema was noted. On the last examination made on June 22, 1954 only residual pleural thickening was evident.

The patient gave a history of having had a severe case of typhoid fever in 1943 while in China. She was hospitalized for six weeks, had positive stool cultures for *E. typhosa*, and had a two month convalescent period. Two weeks after discharge from the hospital, she noted the appearance of a 3 cm. diameter area in the middle of the right leg over the tibia which was swollen, red, hot, and painful. Ten weeks after the onset of her illness, this area began to drain purulent matter. A few weeks later a similar lesion appeared over the left leg. Three to four months later, a lesion which had appeared on the dorsum of the left foot over the fifth metatarsal bone began to drain. Pain was present in both thighs and locally over the bony lesions. The pain was worse at night and almost absent during the day. The lesions cleared without definite therapy and have not recurred.

On physical examination, no asymmetry of the lower extremities and no loss of muscle power or articular mobility was noted. Both tibial shafts were irregularly thickened on palpation over their midportion. There were healed adherent scars over the anterior surface of the tibias and over the left fifth metatarsal bone.

The Kahn test was negative and the remainder of the laboratory findings were normal. A roentgenographic bone survey revealed changes in both tibias and the right femur. The entire thoracic cage, both humeri, left femur, and left foot were normal. The films of the lower extremities showed areas of periosteal proliferation and cortical sclerosis at the middle third of the shaft of the right tibia, 6 cm. in length, and at the junction of the middle and lower thirds of the left tibia, 7 cm. in length (Fig. 1). Similar changes, to a lesser degree, were seen extending over 7 cm. of the proximal third of the right femoral shaft, laterally (Fig. 2). No evidence of nidus or destruction was noted.

The patient's family was not available for study.

DISCUSSION

Keith and Keith[2] in 1926 reviewed the literature on bone involvement in typhoid fever. They noted that in 19,540 cases of osteomyelitis, there were 167 cases due to typhoid, or an incidence of 0.86 per cent. They believed that this figure was low because many cases of typhoid osteomyelitis do not suppurate and complete cure often occurs in the absence of any therapy. These authors noted that occasionally typhoid osteomyelitis appeared as early as thirteen days after the onset of the disease while the latest onset was forty-five years after the acute illness. In the patients having delayed onset, the bone involvement was thought to be secondary to repeated bacteremia from chronically infected foci.

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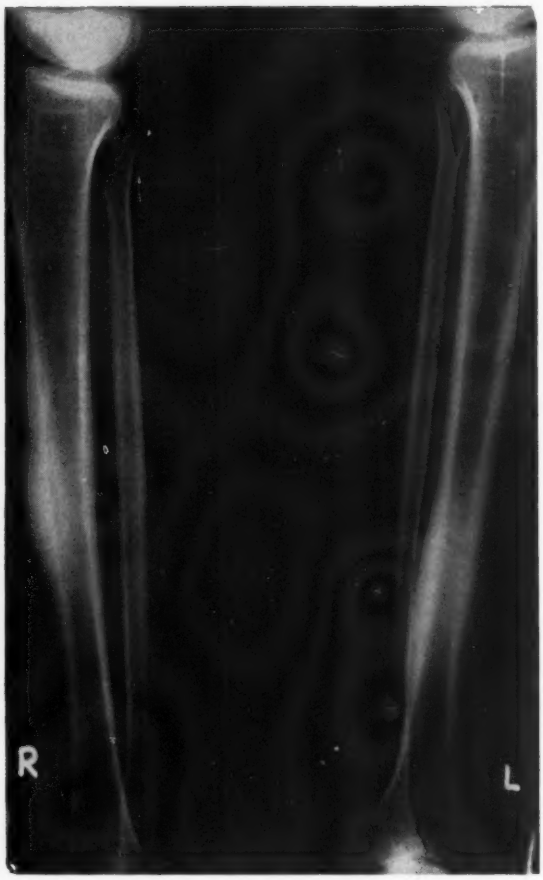

FIG. 1. Lateral roentgenograms of both lower extremities reveal periosteal proliferation and cortical sclerosis at the middle third of the shaft of the right tibia and at the junction of the middle and lower thirds of the left tibia.

Keith and Keith listed the bone involvement as occurring in the following order of frequency: rib, tibia, spine, humerus, ulna, bones of the feet, and clavicle. The periosteum was found to be the part most frequently involved, and the site of involvement was generally in the diaphysis of the long bones, at a greater distance from the epiphysial line than was seen with lesions due to staphylococcal infection.

A report by Dachtler[1] in 1914 on chronic typhoid periostitis described 2 cases with histories and roentgen findings identical with those noted in the case presented above. The author stated that the finding of multiple diaphysial sclerosis secondary to typhoid fever had been seen by other observers. He pointed out that many cases were passed off as due to syphilis, even though, as in his cases, there was no history of syphilis and a negative serology. Dachtler listed the following features as characteristic of chronic typhoid periostitis: (1) periosteal inflammation is most common; (2) multiple lesions are common; (3) bilateral involvement is often seen; (4) tibia is the long bone most frequently involved.

Schinz, *et al.*,[6] recently commented upon personal observations of many cases of diaphysial sclerosis due to late recurrences of typhoid. The cases were recognized because of the appearance of a localized osteomyelitis in long bones and were thought to represent the lighting up of old foci of infection.

DIFFERENTIAL DIAGNOSIS

Several references[4,5] have appeared in the recent radiologic literature to a syndrome characterized by fusiform thickening of portions of the diaphyses of many of the long bones, most frequently the tibias and femurs, usually bilaterally symmetrical. Sclerosis with thickening has also been seen in some of the flat bones, especially at the base of the skull. In the long bones the thickening may be palpable. Six of the 11 cases thus far reported were asymptomatic. When symptoms were noted, they were of a minor nature, such as dull aching, and

FIG. 2. Anteroposterior roentgenogram of both upper femurs shows some periosteal reaction and cortical sclerosis present laterally in the proximal third of the right femoral shaft.

did not incapacitate the patient. The general health of the reported patients remained good. The three groups which have been reported have all been in families, and thus a hereditary factor is suspected.

The etiology of this syndrome is uncertain; there are probably a number of different entities that may lead to these bone changes. Biopsies are inconclusive; in the three cases reported by Ribbing[5] and by Paul,[4] biopsies revealed a nonspecific osteoperiostitis. The diagnosis is based largely on roentgen findings.

As noted previously, Dachtler[1] and Schinz, *et al.*,[6] have reported a number of cases of multiple diaphysial sclerosis in which typhoid was the etiologic factor. The history of draining sinuses occurring early in the convalescence of a known case of typhoid fever makes the diagnosis easy. However, the problem is more difficult in patients who develop chronic foci of infection after an earlier episode of diarrhea and fever which was undiagnosed. Typhoid osteomyelitis may develop long after the acute illness. Also, as Keith and Keith pointed out, many cases of typhoid osteomyelitis go undiagnosed because they heal with only minimal symptoms having been noted and without sinus formation. In the case presented above, the patient experienced mild aching in the thighs and had a residual diaphysial sclerosis in the right femur. If the femur had been the only bone involved, the roentgen study made ten years later would have presented a difficult diagnostic problem. Since typhoid fever was and still is a very widespread disease, it is possible that the 11 previously reported cases of Ribbing's osteosclerosis may have been due to this type of typhoid bone involvement.

Syphilitic periostitis can be ruled out by serologic studies. Roentgenologically, syphilis generally produces a more extensive and more marked osteosclerosis than does typhoid fever. Other authors have listed Caffey's infantile cortical hyperostosis in the differential diagnosis. This can be ruled out firstly, because the patients under discussion are adults, and secondly, because the bone findings undergo progressive clearing in Caffey's disease, whereas they appear to be permanent in typhoid periostitis or Ribbing's osteosclerosis. Engelmann's disease[3] can be ruled out because of its progressive nature as well as the more extensive bone involvement with associated neurologic and muscular findings.

As mentioned previously, a definite differentiation of typhoid periostitis from idiopathic osteosclerosis, as described by Ribbing, is not easily made. It is of interest to speculate on possible interrelations between Ribbing's osteosclerosis and typhoid periostitis. It would seem unlikely for several members of one family to develop osseous complications following an earlier attack of typhoid. Nevertheless, there is still some evidence that the bone involvement in Ribbing's osteosclerosis is secondary to chronic inflammation. Ribbing's case No. 6 developed diaphysial sclerosis in only one site following severe trauma to that area. A biopsy was diagnosed as traumatic periostitis. This case may well be due to chronic inflammation and yet is classed as a case of hereditary diaphysial sclerosis by Ribbing because of the similarity of the roentgen findings to those seen in other members of the family.

There may actually be some relation between the hereditary factor proposed by Ribbing and the appearance of foci of chronic inflammatory disease in bones. In discussing osseous localization of tuberculosis, Schinz, *et al.*,[7] quoted work by Verschner and Diehl, and by Uehlinger, on twins. Sixty to seventy-five per cent of 87 identical pairs of twins were noted to develop osseous tuberculosis in the same sites. This was interpreted as indicating the presence of a gene responsible for hereditary susceptibility of certain bones to infection. Similarly, there may be a hereditary susceptibility to localization of bone reactions in certain of the long bones in Ribbing's disease. Thus, the tendency to multiple diaphysial sclerosis may be an inherited factor which is capable of being stimulated by many agents, *e.g.*, trauma, typhoid, or other inflammatory agents.

SUMMARY AND CONCLUSION

A case of multiple diaphysial sclerosis probably due to typhoid periostitis is presented. The similarity to cases recently reported as Ribbing's osteosclerosis is discussed and possible interrelations of these disease entities are suggested.

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OBSERVATIONS ON PERIARTHRITIS CALCAREA*

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SINCE Duplay's description of the periarthritis scapulo-humeralis syndrome in 1872, no agreement has been reached as to the real concept of this disease. What remained unchanged is that this is a painful condition of the shoulder without any involvement of the joint itself. With the advent of the roentgen ray as a diagnostic tool, it became obvious that in a number of cases this clinical syndrome is associated with calcium salt deposition in the periarticular tissues. It was first thought that the calcium salt deposition primarily develops in the bursa, and the name of subdeltoid bursitis was adopted (Painter,[18] 1907). Codman[3] (1907) showed that it primarily develops in the tendons, and Sandström[20] indicated that periarticular structures other than tendons may also show calcium salt deposition. If a bursa is involved, it is generally secondary to the disease of the tendon. Consequently the terms tendinitis calcarea and peritendinitis calcarea, respectively, were suggested. Recent publications indicate that not only the shoulder, but almost all of the joints of the extremities may be diseased as well. In a previous publication the writer[9] suggested the term periarthritis calcarea (P.C.) for this syndrome. Although in the majority of the cases the primary location of the disease is in the tendon, other periarticular structures such as muscle bundles, tendon sheaths, ligaments, joint capsule and loose connective tissue also may be primarily involved (Sandström and Wahlgren,[22] Copland and Michel,[5] de Lorimier[15] and others). It was, therefore, felt that in the terminology we should not limit ourselves to the tendons and peritendinous structures but should return to Duplay's broader term of periarthritis. This term will be used in the present article, an attempt being made to consider the disease in a systematic review of all of the joints of the extremities. Such an approach provides a better understanding of this entity than separate discussions under the titles of different anatomic areas. The incidence will be dealt with in detail, laying emphasis on less common locations, and cases of thus far unreported locations will be added.

MATERIAL

The observations discussed in this article were made mainly from a five year material (1949–1954) of the Dispensary of the United States Army, The Pentagon. Some material was added from the Civilian Employees Health Service of the Pentagon and from private practice. Whenever statistical methods are used or percentual figures are given, the material was limited to a two-year period (May 1, 1951–May 1, 1953) of the Dispensary of the United States Army, The Pentagon. The material of the latter interval has been systematically and thoroughly reviewed. A diagnosis of periarthritis calcarea was made only in those cases in which the calcification demonstrated by roentgenographic examination was also associated with clinical symptoms and signs. The patients had no other known diseases and there was no history of any intake of vitamin D in excess.

INCIDENCE

In the two-year period mentioned above, the participation of the joints in the order of frequency was found to be as follows: shoulder (59 cases), hip (12 cases), elbow (9 cases), wrist (6 cases without and 8 with involvement of the first carpometacarpal segment), knee (2 cases) and finger (1 case). Every effort has been made to achieve a diagnosis, indicating not only the anatomic region, but also the particular structure (tendon) involved. The location

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of the pain, swelling and redness, the palpatory finding of tenderness or of an occasional mass, and particularly the location of the calcium salt deposition as seen on the roentgenogram, were generally informative in this respect. Passive stretching of the tendon in question was frequently found to be more painful than active contraction of the muscle. This is a common diagnostic sign described in the literature as characteristic of the individual location of the disease. The procedure sometimes cannot be applied in the acute cases because of the severe pain and the generalized spasm present. A forced passive movement, opposite in direction to the physiologic action of the muscle in question, generally precipitates pain in the region where its tendon is inserted. This pain is very marked when contrasted with that precipitated by passive movements in other directions. It is particularly noticed when forced internal rotation of the arm, without or with adduction (behind the back), is used in disease of the rotator cuff, or when external rotation of the arm combined with abduction, is applied in case of involvement of the subscapularis tendon. Forced adduction and internal rotation is probably the proper procedure in involvement of the piriformis, obturator internus and gemelli. In a disease of the gluteus medius tendon it is more helpful to apply active contraction of the muscle. The patient lies on the healthy side and by active elevation of the diseased extremity pain is precipitated on the lateral aspect of the greater trochanter. In the elbow, if the lateral epicondyle of the humerus is involved, forced pronation with the forearm extended and the wrist and hand flexed is the suggested procedure. In disease of the flexor carpi ulnaris tendon forced extension (dorsal flexion) of the wrist followed by radial flexion with fingers extended was found diagnostic by the author. For involvement of the abductor pollicis longus (and extensor pollicis brevis), opposition of the thumb followed by ulnar deviation of the wrist was recommended by Finkelstein.[8]

The procedure aiming to identify the disease in one particular structure is not only of academic interest. It may also be useful in the differential diagnosis in doubtful cases and may indicate that we are really dealing with periarthritis calcarea. The necessity of accurate localization, if any type of surgical therapy is employed, needs no explanation.

DISCUSSION OF THE INDIVIDUAL JOINTS

Shoulder. This anatomic area is extensively discussed in the literature, to which Codman[3] made a classic contribution. A review of painful shoulders in our material —with exclusion of cases where a diagnosis other than periarthritis calcarea could be made—revealed visible calcification on the roentgenogram in nearly 40 per cent of the cases. The site of involvement was as follows:

1. Supraspinatus, infraspinatus and teres minor tendon, commonly referred to as rotator cuff.

2. Subscapularis tendon (Fig. 1.).

3. Lateral to the tip of the coracoid process (Fig. 2), probably the tendons of the coracobrachialis and the short head of the biceps muscle. This location is not uncommon in our experience (4 cases). Such a case was also reported by Key.[11] It generally remains undiscovered since the coracoid process is obscured by other bony structures of the shoulder when the usual technique is used on roentgen examination.

4. Lateral to the acromion process (Fig. 3), probably the deltoid muscle (3 cases).

5. Adjacent to the superior glenoid tuberosity (Fig. 4), probably the long tendon of the biceps muscle (1 case).

6. Adjacent to the inferior glenoid tuberosity (Fig. 5), probably at the insertion of the long head of the triceps muscle (2 cases). It does not change its position in internal and external rotation of the arm and this helps to differentiate it from involvement of the subscapularis tendon.

7. Anterior to the proximal third of the humeral diaphysis (Fig. 6), probably in the teres major tendon. In none of the 3 cases

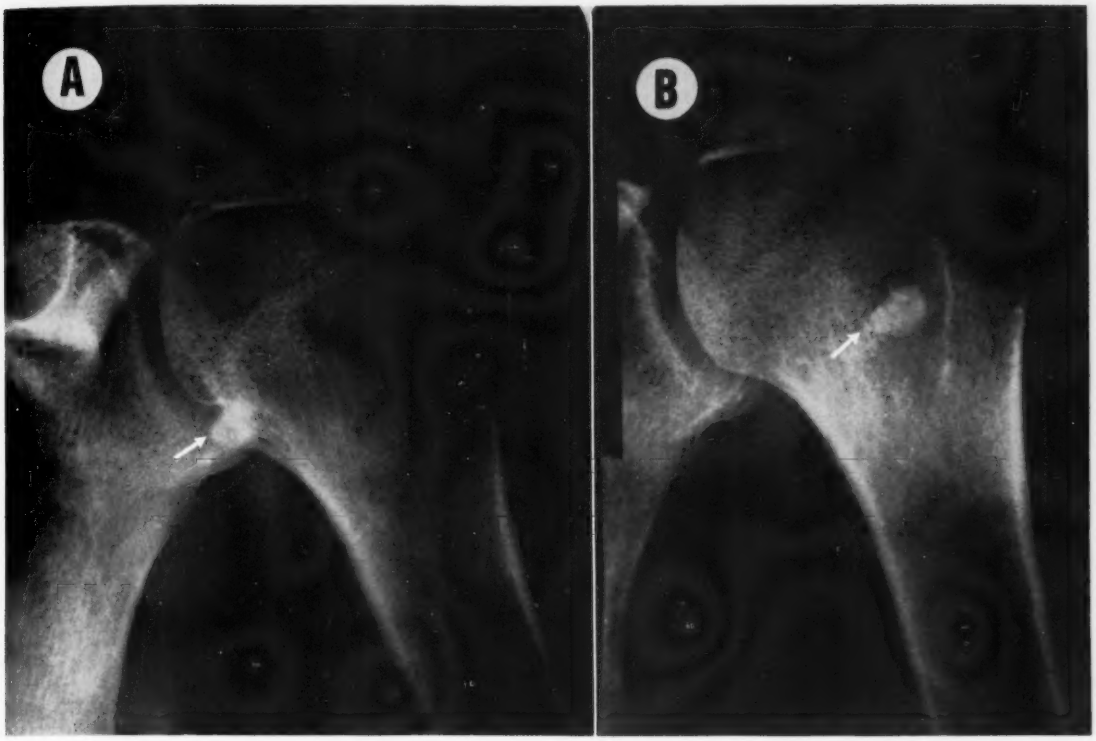

Fig. 1. Calcification in the subscapularis tendon. Roentgenograms were taken with the arm in (*A*) internal and (*B*) external rotation.

observed was any local symptom present. The clinical syndrome resembled that of involvement of the rotator cuff. Though this location is not a rarity, only one similar case was found in the available literature (deLorimier, Moehring and Hannan[16]).

Fig. 2. Calcification adjacent to the coracoid process of the scapula probably in the coracobrachialis and biceps (short head) tendons. There is also calcification in the "rotator cuff" and adjacent to the proximal end of the humerus shaft.

Elbow. This region was affected in 12 cases, the sites being as follows:

1. At the lateral epicondyle (Fig. 7 and 8), area of the common extensor tendon (7 cases). The first such case has been reported by Schmitt[23] (1921). The symptoms and signs are similar to those of the so-called tennis elbow (also called golf elbow, radio-humeral bursitis, epicondylitis or epicondylalgia) and we believe that the terms periarthritis calcarea at the lateral epicondyle and tennis elbow cover identical diseases.

2. At the olecranon process (Fig. 9), triceps tendon (3 cases).

3. At the medial epicondyle, area of the common flexor tendon (1 case). In this case (Fig. 10) the clinical diagnosis was ulnar neuritis. The patient had pain and paresthesia of ulnar distribution; the complaints subsided after one week, but the calcification remained unchanged. In an analysis of this case it should be kept in mind that

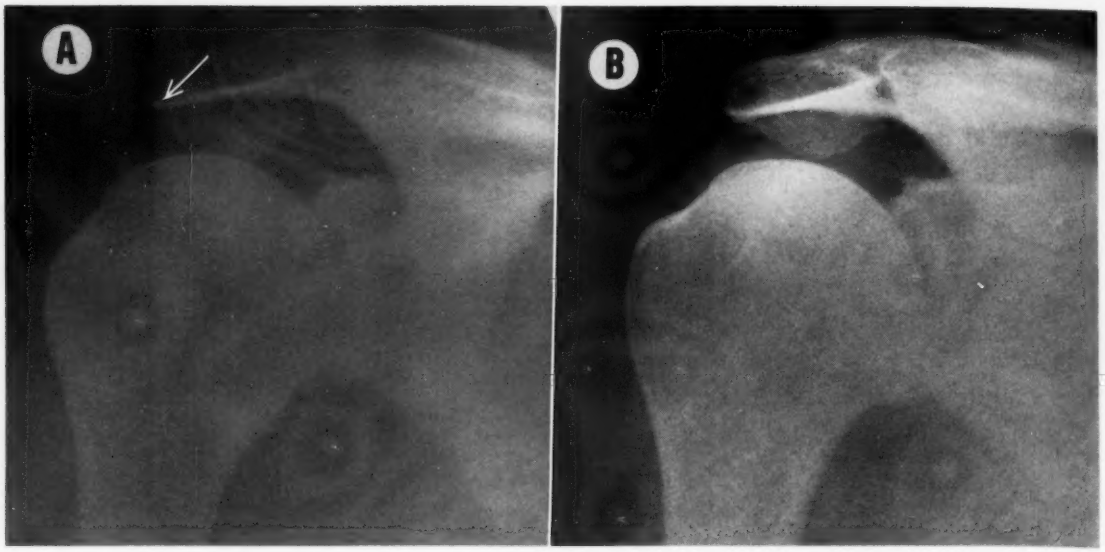

FIG. 3. (*A*) Calcification lateral to the acromion process, probably in the deltoid muscle. (*B*) Six weeks later no calcification is recognizable.

in the area of calcification the ulnar nerve is located between the two heads of the flexor carpi ulnaris muscle. The role of periarthritis calcarea in producing the symptoms of ulnar neuritis is further supported by the spontaneous subsidence of the complaints after a week, which is the usual course of periarthritis calcarea. This condition should always be considered in

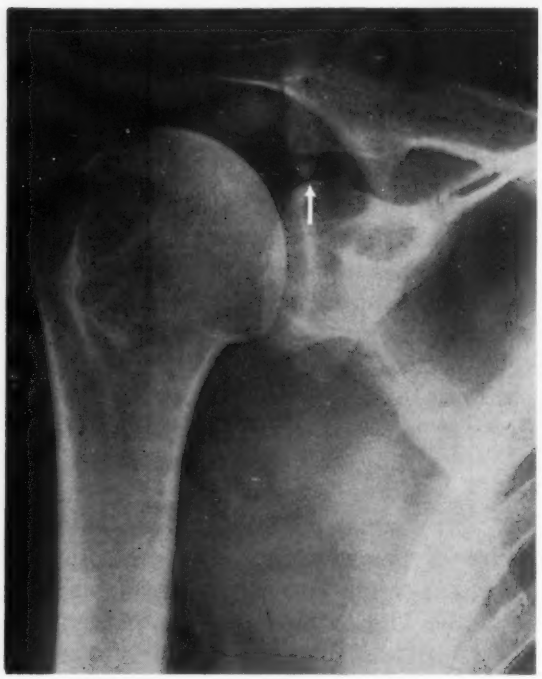

FIG. 4. Calcification adjacent to the superior glenoid tuberosity, probably in the long head of the biceps muscle.

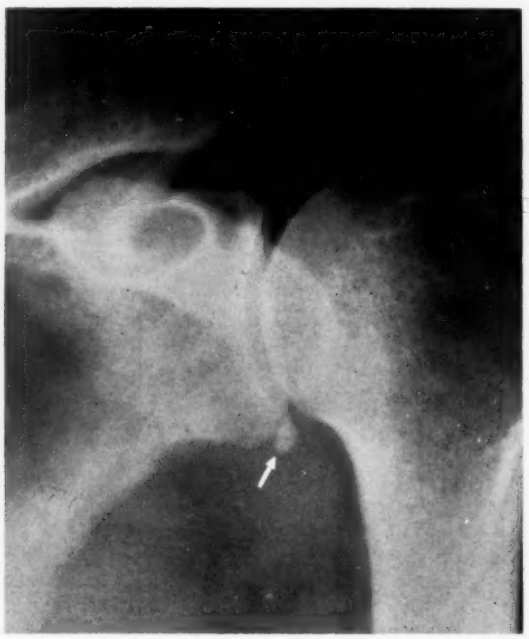

FIG. 5. Calcium salt deposition adjacent to the inferior glenoid tuberosity, probably in the long head of the triceps muscle. The calcific shadow did not change its position after internal and external rotation of the arm. Compare with Figure 1.

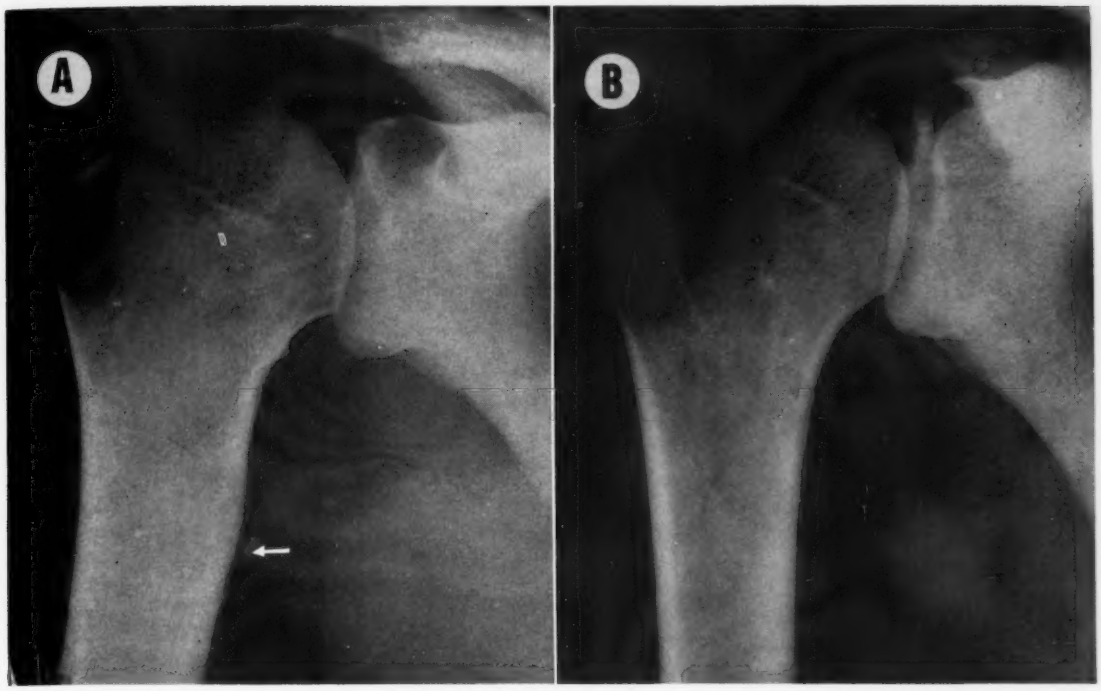

Fig. 6. (*A*) Calcification adjacent to the proximal end of the diaphysis of the humerus. The location of the calcium salt deposition is similar to that seen in Figure 2, anterior to the shaft, probably in the teres major tendon. Figure 2 was taken in external rotation, whereas Figure 6 in internal rotation. (*B*) After disappearance of the calcification.

cases of ulnar neuritis if the etiology is unknown.

Wrist.

1. Flexor carpi ulnaris tendon (Fig. 11). This is the most common location in the wrist (11 cases). The calcification is observed adjacent to the pisiform bone; best visualization can be obtained in the oblique view (pisiform view) with the dorso-ulnar aspect of the wrist on the film.

2. Area of flexor carpi radialis, extensor carpi ulnaris and common flexor tendons, as well as the area adjacent to the medial aspect of the styloid process of the ulna (1 case of each area) (Fig. 12). Two cases of calcification medial to the styloid process of the ulna, similar to our case, were re-

Fig. 7. Calcification adjacent to the lateral epicondyle of the humerus, probably in the common extensor tendon. Patient had symptoms of tennis elbow.

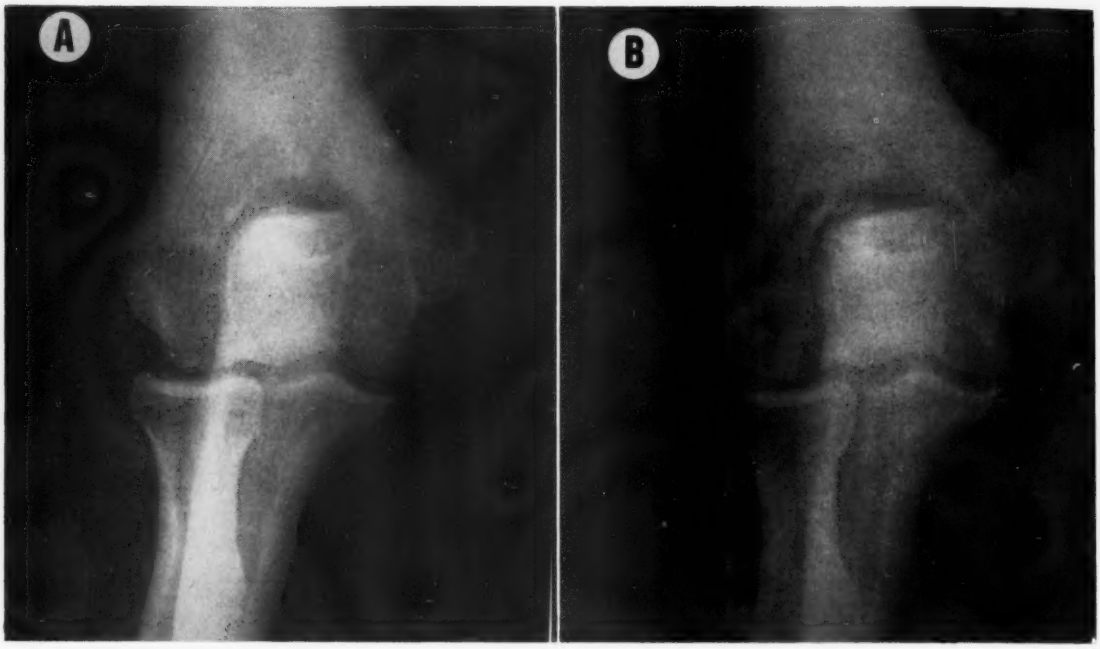

FIG. 8. (*A*) Calcification adjacent to the lateral epicondyle of the humerus. Symptoms of tennis elbow. (*B*) After disappearance of the calcium salt deposition.

ported by Hitchock.[10] In one of his cases, greasy mortar was removed by surgery from the joint capsule. There is no muscle tendon in this area—a fact which is an additional reason in support of the term periarthritis calcarea.

FIG. 9. Involvement of the triceps tendon.

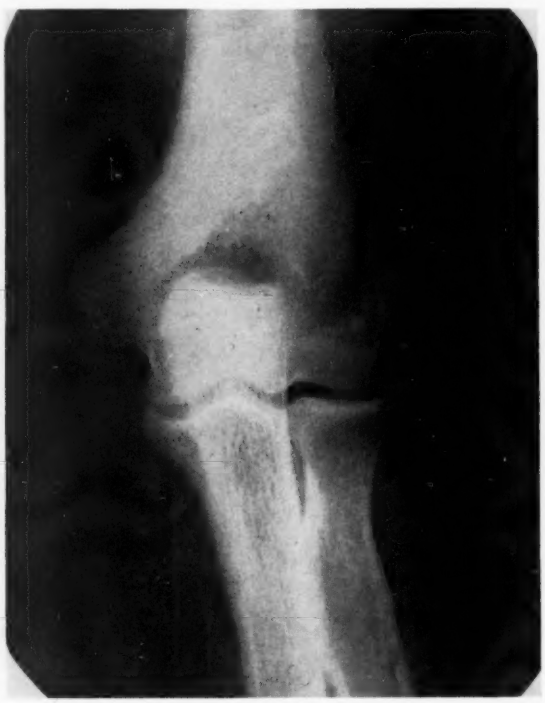

FIG. 10. Calcification adjacent to the medial epicondyle of the humerus. Patient had symptoms of ulnar neuritis.

Fig. 11. Involvement of the flexor carpi ulnaris tendon.

3. Calcification involving the area of the abductor pollicis longus and extensor pollicis brevis tendons topographically may be included in this group, although the anatomy and function of the first carpometacarpal segment are different from the rest of the wrist (Fig. 13). This is the second most frequent involvement in this anatomic area (5 cases). The symptomatology is similar to that of the so-called DeQuervain's disease[7] or, by its descriptive term, stenosing tendovaginitis of the common tendon sheath of the abductor pollicis longus and extensor pollicis brevis muscles. Copland and Michel[5] suggest that this disease belongs to the entity under discussion in this article. This concept seems to be supported by Finkelstein's[8] report of 24 cases; among the 13 cases in which microscopic examinations were performed, calcification was reported in 4 instances. Lamphier, Long and Dennehy,[13] in an analysis of 52 cases of DeQuervain's disease, also found that "calcareous deposits may occur in the sheaths."

Hand. Calcification in the intermetacarpal area (Cooper[4]) and the area of the flexor tendon of the fingers (DePalma,[6] Hitchock,[10] Vasko[27]) has been reported in the literature. In our material there are 2

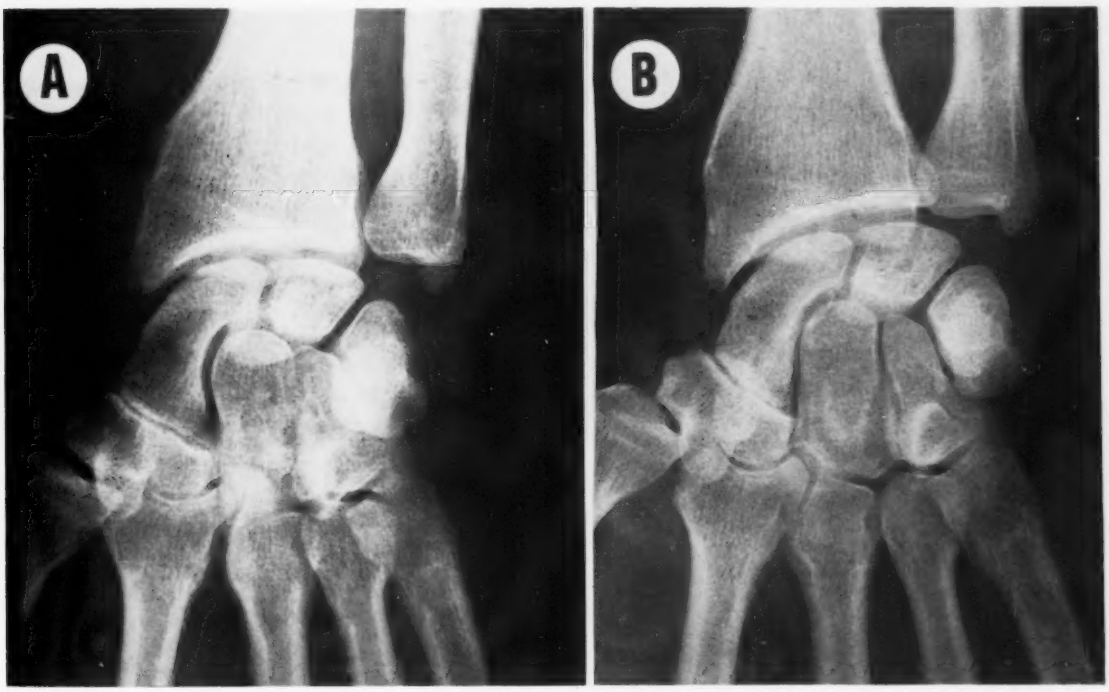

Fig. 12. (*A*) Calcium salt deposition adjacent to the styloid process of the ulna. (*B*) After disappearance of the calcification.

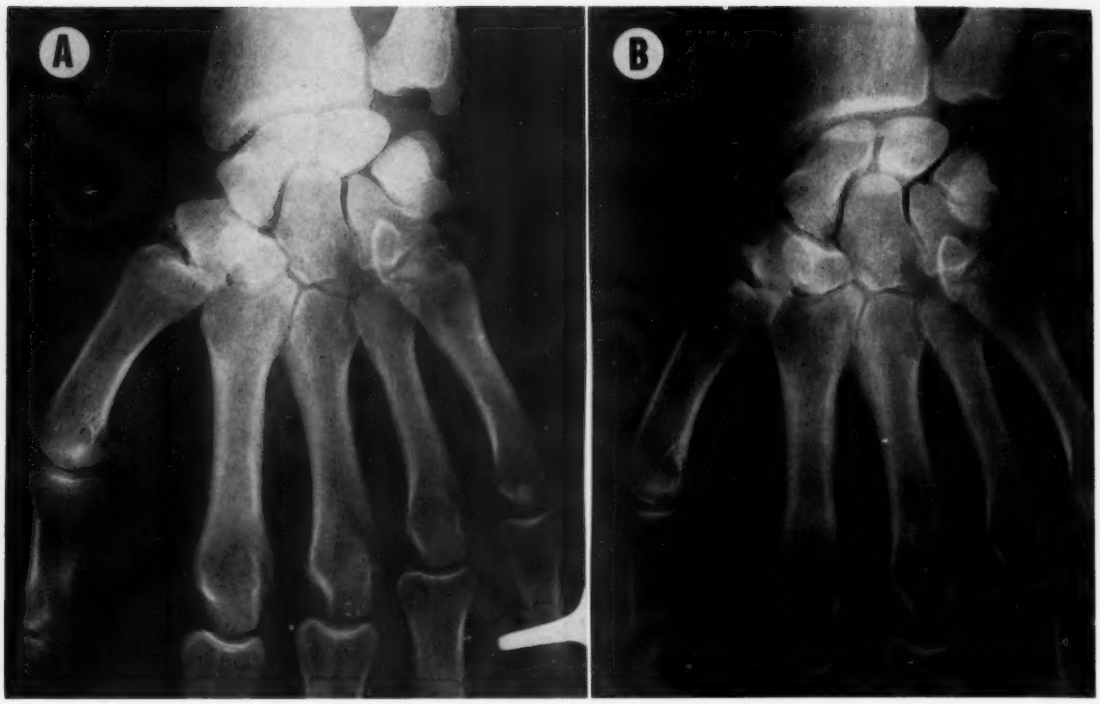

FIG. 13. (*A*) Calcification lateral to the first carpometacarpal joint (probably in the abductor pollicis longus and extensor pollicis brevis tendons). Patient had symptoms of DeQuervain's disease. (*B*) After disappearance of the calcification.

cases with involvement of the intermetacarpal area (Fig. 14), and 2 cases with calcification of the flexor tendon or the joint capsule of the thumb (Fig. 15). We also observed a case with involvement of the extensor tendon of the finger (Fig. 16). No reference to the latter location could be found in the available literature.

Hip. Localization of calcification in the hip is second in frequency and became well known since Stegemann's[26] pertinent report in 1930. Its anatomic sites are:

1. Proximal to the tip of the greater trochanter of the femur (Fig. 17). According to de Lorimier[15] this represents involvement of the tendons of the piriformis, gemelli and obturator internus muscles (9 cases).

2. Lateral to the greater trochanter (Fig. 18), gluteus medius muscle (5 cases).

3. Adjacent to the inferior iliac spine

FIG. 14. Calcification medial to the third metacarpophalangeal joint (probably in the interosseous muscle).

Fig. 15. (*A*) Involvement of the flexor tendon of the thumb (or of the joint capsule). (*B*) After disappearance of the calcification.

(Fig. 19), probably in the rectus femoris muscle (1 case).

4. Adjacent to the upper border of the acetabulum (Fig. 20). We interpreted these cases as involvement of the glenoid labrum (4 cases).

5. Distal to the ischiatic tuberosity (Fig. 21), probably involvement of the adductor magnus muscle (3 cases).

6. Adjacent to the posterolateral aspect of the femur just below the greater trochanter (Fig. 22). This area conforms with that of the tendon of the gluteus maximus muscle (2 cases).

No reference to the last four localizations has been found in the available literature.

Knee. This is an uncommon location. We observed a case with calcification just proximal to the head of the fibula (Fig. 23). This is the area of the insertion of the biceps femoris muscle. Calcification at the medial epicondyle has been reported (Lamb,[12] Key[11]). In answer to the question as to whether or not this localization of the disease is identical with Pellegrini Stieda's disease as suggested, we have insufficient data to form an opinion. In the literature, fracture, subperiosteal hematoma, myositis ossificans, post-traumatic ossification of the internal ligament, etc., have been made responsible for the abnormal bone formation adjacent to the medial condyle of the femur. Involvement of the quadriceps tendon (Hitchock[10]) and calcification at the lateral epicondyle of the femur (Brumbaugh[2]) are also reported.

Ankle and Foot. A case with calcification behind the medial malleolus was also observed (Fig. 24). This thirty-two year old

FIG. 16. (*A*) Involvement of the extensor tendon of the finger.
(*B*) After disappearance of the calcification.

white male has had intermittent pain and swelling in the left ankle. Nine years previously, arthrotomy had been performed and "calcium deposits were removed." Nevertheless the pain later recurred. Recently the patient also developed pain in the right shoulder and subsequently calcification was found in the area of the supraspinatus tendon. Roentgen examination of the left ankle at this time revealed a rounded small calcific density distal and posterior to the medial malleolus. This density was homogeneous and its appearance was similar to that commonly seen in calcification of the rotator cuff of the shoulder. Though a diagnosis of osteochondritic body was made elsewhere, there can be no doubt that the patient has had periarthritis calcarea of the left ankle with involvement of one of the flexor tendons or of their sheaths. Calcification lateral to the lateral malleolus (Hitchock[10]) and lateral to the calcaneocuboidal articulation (Miller[17]) was also reported.

Spine. In a recent article, Sandström[21] calls attention again to calcification in the intervertebral disks. He describes 2 cases with calcification in the central portion of

Fig. 17. Calcification in the common tendon of the piriformis, obturator internus and gemelli muscles.

the disks of the dorsal and lumbar spine. The finding was associated with back pain in the respective segment. After roentgen therapy the pain subsided and the calcification became smaller or disappeared. Sandström grouped these cases in the syndrome under discussion. Silverman,[25] in a report on 7 cases of calcification of the intervertebral disks in childhood, expresses doubt as to any relationship between pain and such calcification. A comprehensive study of the intervertebral calcification had been earlier reported by Schmorl and Junghanns[24] who considered this type of calcification degenerative in nature and did not attach any clinical significance to it.

PARALLELISM OF FUNCTION AND DISEASE

The etiology and pathogenesis of periarthritis calcarea are obscure. Though the majority of the authors believe that trauma has a role in the etiology, this still remains a controversial issue. More frequent incidence (in the upper extremity) has been reported on the right side as compared with the left, which supports trauma as an etiologic factor (Bosworth,[1] Cooper[4]). In our material the incidence is about equal in the two sides both of the upper and

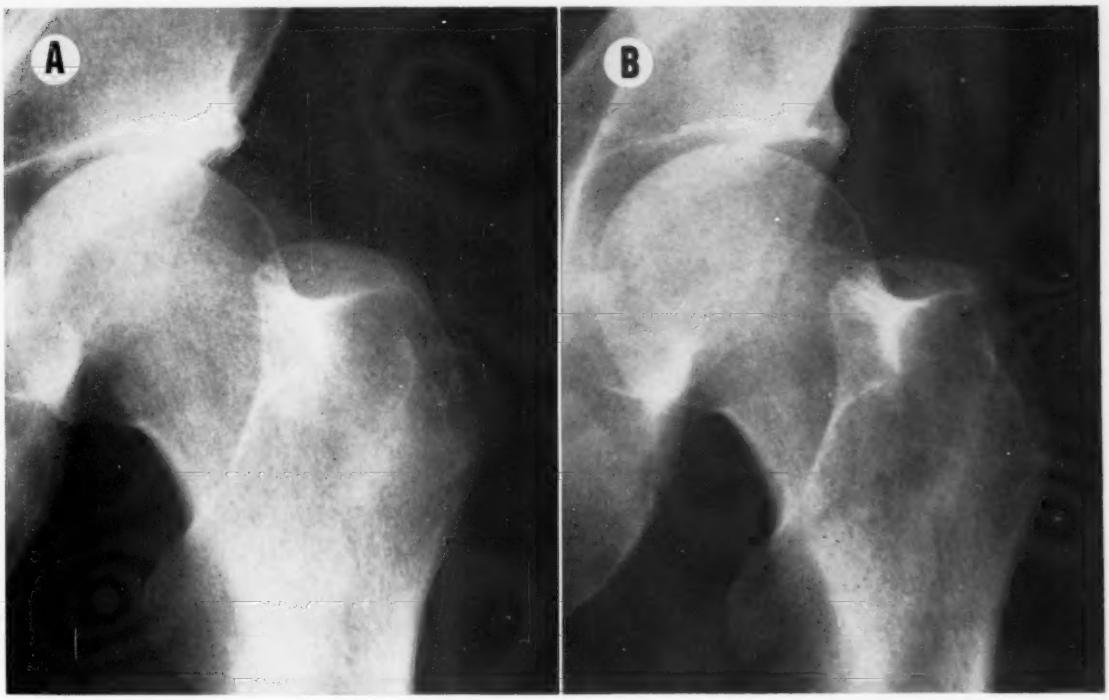

Fig. 18. (*A*) Calcification lateral to the greater trochanter of the femur (gluteus medius muscle). (*B*) After disappearance of the calcification.

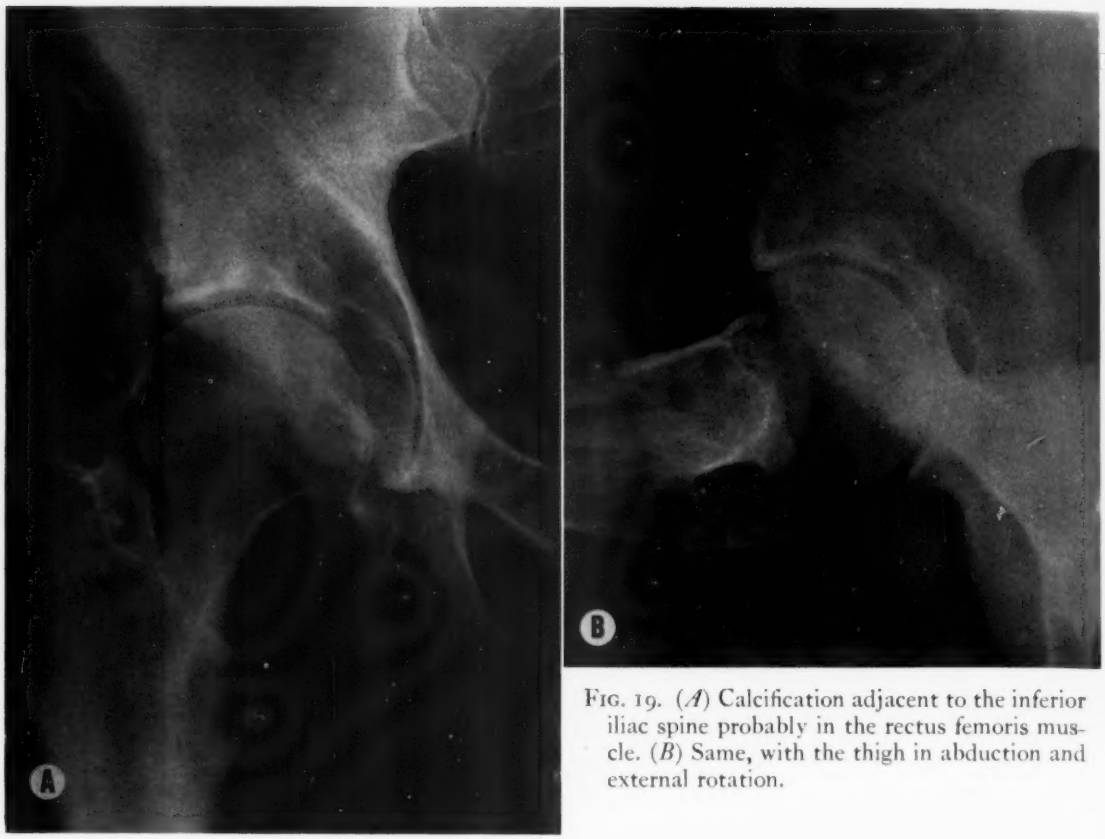

Fig. 19. (*A*) Calcification adjacent to the inferior iliac spine probably in the rectus femoris muscle. (*B*) Same, with the thigh in abduction and external rotation.

Fig. 20. Calcium salt deposition adjacent to the superior border of the acetabulum (probably in the glenoid labrum).

lower extremities. In an attempt to better understand this disease, it seemed of interest to know whether or not there is any relationship between the range of motion and the incidence of periarthritis calcarea in the different joints. Such an investigation was carried out in a systematic review of the extremities.

The common function of the joints are flexion, extension, abduction, adduction, rotation and circumduction. The latter is not considered in this computation, because actually it represents only a combination of other movements. The range of motion is expressed as the mathematic summation of all of the principal movements around a joint (Table 1).

The left column lists the joints in the order of range of motion. An attempt was made to express the range of motion in mathematic form, and the figures indicate

Fig. 21. (*A*) Calcification distal to the ischiatic tuberosity (probably in the biceps femoris muscle). (*B*) After the disappearance of the calcification.

Fig. 22. Calcification adjacent to the posterior aspect of the femur just distal to the greater trochanter (probably in the gluteus maximus tendon).

Fig. 23. Calcification proximal to the head of the fibula, probably in the biceps femoris muscle.

FIG. 24. (*A*) Calcification adjacent to the tip of the medial malleolus. (*B*) Same, in slight internal rotation. (See text for history.)

the approximate summation of the motions obtained mainly from data by goniometry (Pullen[19]).

The column commences with the shoulder, a ball and socket joint (gynglimus). This is the most movable joint of the extremities, and besides it has additional movements of the auxiliary joints and of the scapula. The hip has a grossly similar anatomic arrangement and similar types of motion, but its range is considerably less. Next in order is the elbow, which in addition to flexion and extension has a specific function of pronation and supination. The fourth location is occupied by the wrist, if the first carpometacarpal segment (area of the abductor pollicis longus and extensor pollicis brevis tendon) is not con-

TABLE I

Range of Motion		Incidence of Periarthritis Calcarea (in the present material)	
1. Shoulder (scapulo-humeral joint+movements of the auxiliary joints and of the scapula)	500	1. Shoulder	69%
2. Hip	310	2. Hip	12%
3. Elbow	250	3. Elbow	10%
4. Wrist, without the first carpometacarpal segment	205	4. Wrist, without the first carpometacarpal segment	7%
4*a*. Wrist, with the first carpometacarpal segment	255	4*a*. Wrist, with the first carpometacarpal segment	9%
5. Knee	145	5. Knee	2%

sidered. Though this segment is different in its anatomy and function from the rest of the wrist, topographically it is considered as a part of it. Fifth in order is the knee. The latter has not only flexion and extension movements, but also slight adduction and abduction as well—when the knee is flexed. The rest of the joints with a significant range are essentially hinge joints with extension and flexion only, their range being less than those listed above.

In the right column the joints are grouped according to the incidence of periarthritis calcarea. The respective figures on the right indicate the frequency of involvement in a two year period at the Dispensary of the United States Army, The Pentagon. The two columns are essentially identical. The only discrepancy is in the wrist—if the first carpometacarpal segment is included. The discrepancy is, however, minimal and materially does not change the gross parallelism of the two columns. The considerable difference in the mobility of the shoulder and other joints is reflected by an excess of percentage in the incidence of periarthritis calcarea in the shoulder. It is apparent from this compilation that the frequency of periarthritis calcarea grossly parallels the range of motion of the joints.

This parallelism becomes more apparent when one studies the motion of the elbow more thoroughly. The elbow is composed of two different joints. The trochleo-humeral joint on the medial side is a hinge joint with flexion and extension movements. The radio-humeral joint on the lateral aspect has—beside flexion and extension—a special function, namely, pronation and supination. This excess of motion on the lateral side results in a much higher incidence of the disease at the lateral epicondyle as compared with the medial; the proportion is 7 to 1 in our material. The difference in the anatomy and function of the two joints acquires greater significance in the etiology if we consider the similarity of the bony anatomy of both epicondyles of the humerus and the grossly similar anatomic arrangement of the muscles in their immediate vicinity.

The observation indicated in these two columns, that the disease of different joints can be subjected to a general rule, seems to support the prevailing present concept that we are really dealing with one entity, differing only in location. In the question of etiology, it seems probable that there is a causative relationship between the motion and the disease. In other words, this observation supports the concept that trauma, though not qualified, has a role in the etiology.

SUMMARY

The term periarthritis calcarea is suggested for the disease known as bursitis, tendinitis or peritendinitis calcarea because various periarticular structures may be involved. The disease is discussed in a systematic review of all of the joints of the extremities, and thus far unreported localizations of periarthritis calcarea have been added. The observation is made that there is a gross parallelism between the incidence of this disease and the physiologic range of motion of the joints.

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HYPERCHOLESTEREMIC XANTHOMATA OF THE TENDONS

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IN AN excellent classification of the lipidoses by Thannhauser,[3] the xanthomatoses are divided into hypercholesteremic, hyperlipemic and normocholesteremic groupings. The hypercholesteremic xanthomatoses are of two varieties, the familial and those secondary to liver disease. The familial hypercholesteremic xanthomatoses clinically become evident in one or more of the following manifestations: (1) xanthelasma of the eyelids; (2) cutaneous xanthomata of the papular and/or flat varieties (xanthoma tuberosum et planum) (3) xanthomata of the tendons; (4) xanthomata of the blood vessels and endocardium; (5) "forme fruste," a group with hypercholesteremia as the only objective clinical manifestation, but in which the patients exhibit anginal episodes and coronary thrombosis.

The only manifestation that the radiologist is apt to become concerned with in these familial hypercholesteremic xanthomatoses is the tendon xanthoma. This entity presents a consistent clinical picture, but, because it is not very common, most radiologists are not familiar with it. In the American radiologic literature one case was reported in 1920; no other reference was found except a few abstracts of case reports in the foreign literature.

In 1920, Merrill[1] reported a case showing some small cortical erosions of the phalanges of the hands and feet in a twenty-seven year old white woman who had multiple tendon xanthomata of the extremities associated with hypercholesteremia. One of the foreign reports was by Schinz,[2] who in 1934 reported the case of a fifty-two year old man with tendon xanthomata of the fingers and punched-out defects and erosive changes of some of the bony phalanges.

Tendon xanthomata tend to occur on the extensor surfaces. They vary in size from a few millimeters to several centimeters. They are particularly prone to occur on the dorsal aspects of the fingers and knuckles, and very characteristically involve the Achilles tendons in a nodular cylindrical fashion, producing a marked thickening that is seen clinically as a vertical fullness. This appears on the roentgenogram as a sharply defined thickening of the Achilles tendon, easily distinguished by the contrast of the fat pad anterior to it. The nodules may occur near the knees and near the points of the elbows. They are firm and relatively nontender. The overlying skin is not discolored and is movable over the tumors. When the nodules are very large, small focal softening may rarely occur, but they usually do not ulcerate or drain. Histologically, these nodules contain a few giant cells and characteristically show the foam cells that contain cholesterol. Cholesterol crystals can be demonstrated in softened parts of the xanthomata.

This condition is a familial one, and any of the forms listed above can be found singly or in combinations in a number of the members of a family, though some members may not be affected. The elevated blood cholesterol levels are the common denominator in a family. The cause of this metabolic disturbance is, of course, not known.

The radiologist may be called upon to make soft tissue roentgenograms of the tendon nodules. These are fairly well circumscribed and have aqueous tissue density rather than fat density. In some of these patients there may also be found destructive bone lesions caused by pressure erosion and slight local infiltration. These bone lesions are commonest in the small bones of the hands. They show no perifocal

FIG. 1 and 2. Case 1. There are numerous well-defined punched-out areas of cortical erosion associated with the xanthomata of the extensor tendons of the hands.

FIG. 3 and 4. Case 1. Bone defects similar to those in the hands are present in the feet.

Fig. 5 and 6. Case 1. The greatly thickened Achilles tendons are sharply demarcated by the anterior fat layer. They are 4 to 5 times the normal diameter.

Fig. 7 and 8. Case 1. Clinical appearance of the soft tissue masses on the hands and the greatly thickened Achilles tendons.

FIG. 9. Case I. Tendon xanthoma at point of elbow. No bone involvement.

osseous reaction but may exhibit a fine cortical margination. They tend to involve the surface of a bone but, if seen face-on, the bone lesions may seem to be within the medullary cavity. They are rounded or oval and punched-out, and are in close juxtaposition to a tendon nodule. These bone defects associated with soft tissue masses resemble closely the picture in gout, and, indeed, when the clinical aspects are not known these patients are usually misdiagnosed as gout. Neurofibromatosis may also be considered in the differential diagnosis. The bone defects in tendon xanthomatosis, though they are often close to joints, show some tendency not to involve the articular margins of the joints as much as do the ones in gout. This tendency may help the radiologist differentiate these two conditions. Bilateral involvement of the Achilles tendons should offer a strong lead to the correct diagnosis.

A similar terminology may cause confusion with xanthomata or giant cell tumors of the tendon sheath, or, as Thannhauser[3] terms them, "single xanthomat-

FIG. 10 and 11. Case II. Greatly thickened Achilles tendons by the xanthomatous involvement are readily discernible.

ous giant cell tumors." These are not part of a systemic disease, as are the hypercholesteremic tendon xanthomata, but are single lesions, 1 to 3 cm. in diameter, lobulated, well encapsulated, and almost always found distal to the wrists and ankles. More common in females than males, they tend to involve the flexor surfaces, whereas the hypercholesteremic tendon xanthomata tend to involve the extensor surfaces. While the familial multiple condition may appear in childhood, the single giant cell tumor of the tendon sheath usually appears in adulthood or middle age. The blood cholesterol level is normal. Included in the rather cellular histologic picture are giant cells, phagocytic cells and some foam cells.

REPORT OF CASES

Members of two families manifesting hypercholesteremic tendon xanthomata were observed by the authors.

Case I. W. S. A thirty-four year old white male was admitted to the Cooper Hospital on December 22, 1954, with the diagnosis of rheumatic heart disease and subacute bacterial endocarditis. Examination revealed aortic stenosis and mitral insufficiency. Blood culture showed Staphylococcus aureus. The patient was found to have large, soft pedunculated masses, yellowish in color, on both elbows and buttocks and extensor tendons of the fingers. In addition, there were masses along both Achilles tendons. Removal of three of the lesions showed benign xanthomata containing cholesterol crystals. Serum cholesterol averaged 500 mg. per cent.

Questioning the patient revealed that he had a maternal aunt with the same condition, one sister has xanthelasma, and a maternal aunt who died at the age of forty-eight with a coronary occlusion also had xanthomata.

Figures 1 and 2 show the soft tissue tendon masses in the hands and the associated well-defined punched-out bone defects with slight marginal cortication. Similar bone defects are noted in the feet in Figures 3 and 4. The marked involvement of the Achilles tendons is shown in Figures 5 and 6. The clinical appearance of the hands and ankles is pictured in Figures 7 and 8. A tendon xanthoma without associated bone defect is shown on the extensor surface of the elbow in Figure 9. Similar masses were present on the anterior aspect of the knees and on the buttocks.

Case II. K. M. A sixty-seven year old Jewish woman had had swellings at the backs of both ankles and lower legs for thirty years. These swellings were at first painful but soon became relatively painless. Over the course of many years nodules also appeared on the extensor tendons of both hands. Roentgenograms of the tumors of the Achilles tendons are shown in Figures 10 and 11. A tumor at the left elbow is shown in Figure 12. The blood cholesterol level was 490 mg. per cent. Roentgenograms of the hands showed no bone lesions associated with the relatively small tumors.

This woman has one son with extensive xanthomata of the hands. Two daughters show hypercholesteremia and one has xanthelasma palpebrarum and a small tendon xanthoma on the hand. The daughter of the latter is presented as Case III.

Fig. 12. Case II. Tendon xanthoma near point of elbow.

FIG. 13 and 14. Case III. Thickened Achilles tendons in an early case. The diameter is slightly over twice normal.

CASE III. G. S. A sixteen year old female, a granddaughter of Case II, had noted some "lumps" for about a year on her hands and the backs of the ankles. The latter were somewhat painful at the onset. The rather small nodules on the hands were in the extensor tendons. Roentgenograms showed no bone lesions. Figures 13 and 14 show the early xanthomatous involvement of the Achilles tendons, which are at least twice as thick as normally. The blood cholesterol level was 500 mg. per cent.

SUMMARY

Hypercholesteremic xanthomata of the tendons is one manifestation of hypercholesteremic xanthomatosis, a familial metabolic disturbance. The tumors are found on extensor surfaces, especially the hands, feet and Achilles tendons. In addition to the soft tissue masses which the radiologist may encounter, there may occur punched-out osseous defects in association with these masses, presenting a gout-like or neurofibromatosis-like picture. The tendency to spare the articular margins of the joints and the involvement of the Achilles tendons bilaterally would help distinguish it from gout roentgenologically. Three cases are presented from two families with this condition.

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ROENTGENOLOGIC ASPECTS OF MOTOR VEHICLE ACCIDENTS

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ST. JOSEPH, MISSOURI

THE medical aspects of motor vehicle accidents involve considerations of factors governing normal and abnormal phases of motor transport. The latter includes preclinical, paraclinical and post-clinical elements in addition to the clinical ones. It means accident prevention, physical factors in injuries, aids to recovery, and ultimate rehabilitation. Radiology pervades this entire field and interlocks with every facet of it. The domain of the radiologist in motor vehicle accidents is a vast one, therefore. I shall try to present its integrated clinical life history (Fig. 1). For reasons which will become apparent, ideas as well as facts and methods will be mentioned. Also, the subject entails a sense of the cultural and humanitarian interests of physicians everywhere and of whatever branch they practice and represent.

PRECLINICAL: ACCIDENT PREVENTION

Aside from the mechanical factors involved, the human element in the causation of accidents is conceded to be the most difficult to control. Epitomized, motorist safety is dependent upon driver proficiency, good highway facility, reliable performance of the vehicle, and, what is seldom clearly understood, the real and imagined protection offered by the vehicle under crash conditions. There are no valid data which connect, for example, physical deficiencies with greater-than-chance accidents. There are no uniform minimum medical standards for driver licensure in this country (Fig. 2). Moreover, studies purporting to analyze the accident records of those with physical defects tend to indicate that these drivers' records compare favorably with the rest of the driving population. However, the

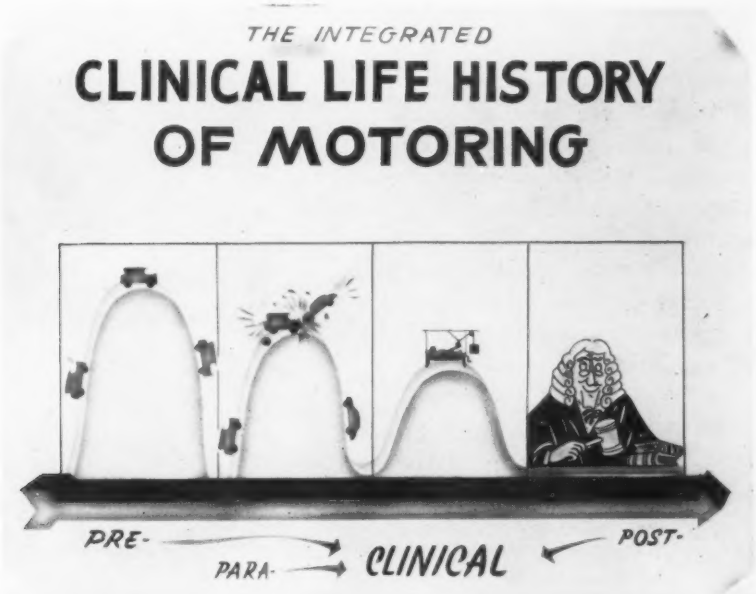


FIG. 1. Clinical frontiers are expanding in accordance with epidemiologic principles, *i.e.*, considering interactions between agent (vehicle), host (rider), and environment (external and internal automotive ones).

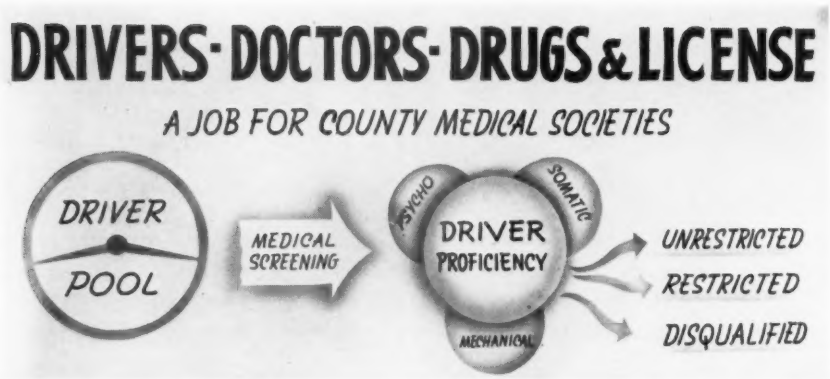
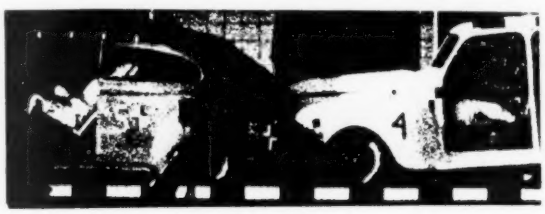


FIG. 2. Medical screening for driver licensure should begin on a voluntary basis until it is made legally compulsory. People have been conditioned to this kind of thing through pre-employment, job fitness, and armed forces medical examinations. The project needs the support of physicians and industry.

realization that the latter contains the very elements we might wish to eliminate or restrict in drivers more or less cancels out these conclusions. Thus, the surface has hardly been scratched along this line. In the meantime, exploration of possibilities and probabilities yields some interesting ideas. More recently I have evidence to show that drivers with cervical arthritis are more prone to rear-end and angular impacts—especially at intersections.

One of these may be deduced from the high incidence of varying degrees of cervical arthritis in drivers (and passengers) who become involved in whip-lashing injuries of the neck. The vast majority of these accidents occur at intersections from rear-end and/or angular vehicular collisions (Fig. 3). It is conceivable that the rotatory and rocker action of the cervical spine which is needed to meet these danger cues is inadequate in some of these drivers, *i.e.*, movements of the neck to supplement the field of vision peripherally. The implications of this statement are further strengthened with respect to those cases which are associated with neck-shoulder-arm pain and disability. Even more credence would be given the above assumption if it could be proved in order that such arthritic drivers be made more liable legally in courts of law than normal ones. This indirect evidence is difficult to get because most of these personal injury cases are settled out of court.

Be that as it may, the radiologist (master of living morbidity) has made a beginning toward medical screening for driver licen-



(a)

(b)

REAR-END COLLISION

(a) 125 MILLISECONDS AFTER CONTACT

(b) 200 MILLISECONDS AFTER CONTACT

FIG. 3. Experimental reproduction of the classic bodily reaction to forces from rear-end collisions. (Supplied [and performed] by Severy and Mathewson of the Institute of Transportation and Traffic Engineering, University of California at Los Angeles.)

sure by his painstaking observations and reports to his colleagues with respect to both primary and *accessory* diagnoses. The latter has also served to help classify the acute and chronic sprains of the neck from motor vehicle accidents. Since the vast majority of cases with cervical arthritis obviously had this condition prior to their alleged accidents (as shown on the roentgenograms), this was looked upon as a complication rather than a result of the accident. In other works, in the absence of subluxation, dislocation, or fracture (vertebral body or posterior elements), these cases were considered to be sprains primarily when this picture was substantiated by the history and physical examination. These two advances—categories in accident prevention and classification of injuries—may also be applied by inference to other conditions, diseases and congenital anomalies.

It is well to point out in this regard that fissure fractures of the posterior elements of the cervical spine are frequently difficult to differentiate from congenital anomalies, but must not be overlooked. Already, the medicolegal implications are piling up. Some of these complicating factors are cleared up in the presence of positive roentgenographic evidences of skeletal injury, but not all of them. Other implications with respect to cause and effect also crop up. These underscore the natural evolutionary interrelationships between the various phases of motor transport.

For example, a single situation like cervical arthritis (or similar condition) may initiate a chain of events all along the line in motor transport. Thus, in addition to increasing the chances for accidents (?), the affected individual is definitely made more vulnerable to whip-lashing forces. This in turn calls for ever increasingly larger factors of built-in automotive safety design because cervical arthritis appears to be a common ailment among the motoring public which becomes involved in such injuries (Fig. 4). Also, in these people, the clinical features of such injuries are intensified, reflecting in resistance to treatment and tendency toward chronicity (in both non-litigating and litigating patients). Finally, arthritis and other accessory physical aberrations further complicate the medicolegal considerations because the issue of aggravating conditions existing prior to the accident is always raised. Not infrequently, especially in cases which have dragged on for years or in which the arthritis is minimal, the more direct causal relationship of injury to arthritis is challenged.

In all of these matters, the radiologist is in the enviable position of one who is allowed to take a solid objective attitude, and rightly so. More than that, his observations on the physical situation of motorists in general and drivers in particular bring the necessity for driver medical screening into sharp relief. Nothing can supersede accident prevention anyway. All

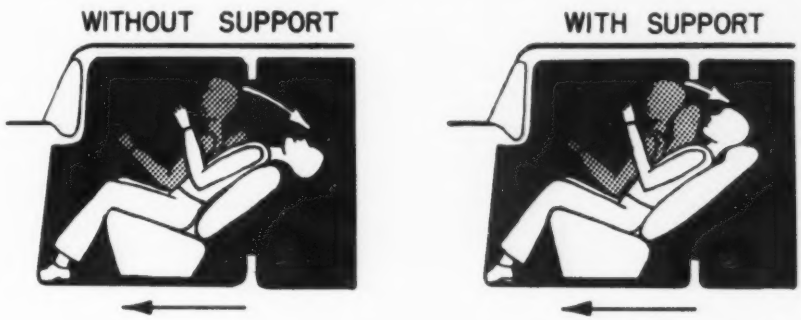


Fig. 4. Showing the attenuating effect of a head rest during whip-lashing accidents (Severy and Mathewson).

other aspects of the problems are supplementary to this concept of primary safety. However, the epidemic number of accidental injuries and deaths, despite the lowering accident rate, recalls the necessity for reducing injuries under crash and/or upset conditions in its own right. This means a better understanding of the physical factors which govern accidents and injuries, *i. e.*, the etiology of motor vehicle accident injuries.

PARACLINICAL: PHYSICAL FACTORS IN ACCIDENTAL INJURIES

In these days of vicarious criticism of the motor vehicle, it is forgotten that its structural elements do afford a certain amount of protection under crash conditions. It is for this reason that we have four basic subgroups of motorist casualties: those who escape injury in accidents fatal to others; those who die outright; those who are injured but recover; and those who are injured and die later from complications attending their injuries. However, prior to accidents, there is a tendency for some drivers and riders to assume an imaginary protection which does not exist. This reaction or failure to react to what seem to be obvious danger cues has been related to the relatively recently experienced motoring dangers. In other words, some persons—who might cringe from a mouse (age old experience)—have not yet acquired a sense of the dangerousness of some aspects of motoring; for example, speeding beyond the limits of safety. This is the status quo until there is a change in the relative velocity between the rider and his immediate environment. When this happens such factors as magnitude of force, direction of impact, duration of force application or pressure, velocity at onset of impact, area of the body impacted and square surface area affected will determine the results. Above all, survival calls for intactness of the rider compartment—at least that in his immediate vicinity.

In a general way, the configuration of motor vehicle accidents reflects physical laws of time, space and motion, *i.e.*, inertia, deceleration and/or acceleration and dissipation of kinetic energy. The basic interactions involve principles of force exchange (Fig. 5). In other words, it becomes a matter of absorption of energy. Structure does it most dramatically on the outside (except in cases of forcible ejection of riders). This is manifested by deformation and collapse of the vehicle. On the inside, unfortunately, absorption of energy by the human body results in injury and/or death. Therefore, the greater the deformation on the outside, the longer the decelerative distance, and the lesser the magnitude of the decelerative rates and forces (g's) (Fig. 6).

A properly suspended rider will then, in the presence of an intact cabin, also go the whole distance with the undamaged portion of the vehicle. His jolt will be diminished in accordance with the roll-with-the-punch principle. Tenets of automotive engineering safety dictate that materials and design should allow maximum deformation control or crumple rates.

Forcible ejection from a moving vehicle illustrates a special case (Fig. 7). Under these circumstances the forces which might act upon such individuals are wholly unpredictable. Crushing forces are to be expected when the ejected person is run over by his own or another vehicle. When this happens, drivers are prone in panic to brake and bring their vehicles to a *deadly stop on top of these unfortunates*. Such crushing forces may be ameliorated by going over as rapidly as possible instead.

On the inside the forces acting on freely moving occupants under crash conditions are chiefly applied ones or impulsive from impacts onto interior design (Fig. 8). The front part of the compartment (windshield, dash and steering controls) present varying injury potentials. The pattern here has been studied from the crash-impact engineering point of view. From this was evolved the premise that source of injury (impact, seating) was an important factor to be eliminated through safety automo-

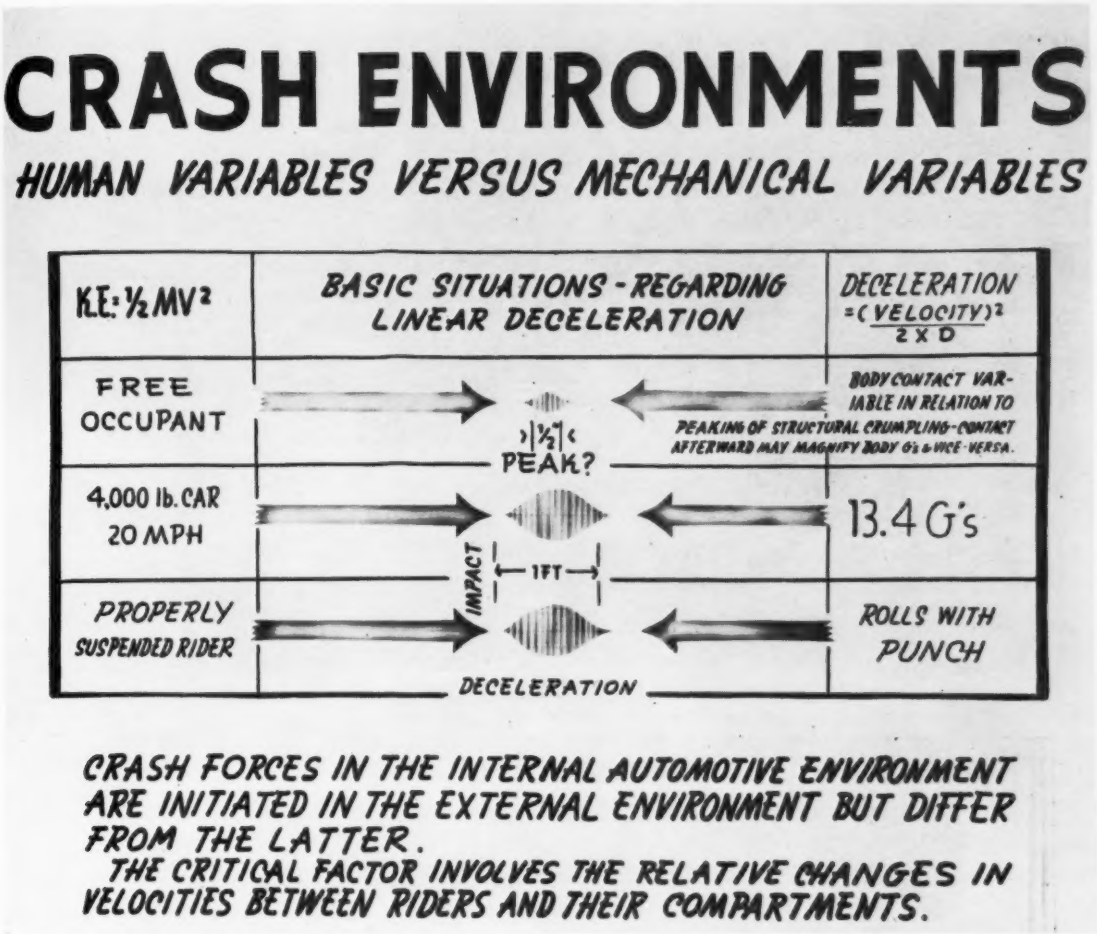

CRASH ENVIRONMENTS
HUMAN VARIABLES VERSUS MECHANICAL VARIABLES
K.E.=½MV²
BASIC SITUATIONS - REGARDING LINEAR DECELERATION
DECELERATION =(VELOCITY)² / 2 X D
FREE OCCUPANT
BODY CONTACT VARIABLE IN RELATION TO PEAKING OF STRUCTURAL CRUMPLING-CONTACT AFTERWARD MAY MAGNIFY BODY G's & VICE-VERSA.
PEAK?
4,000 lb. CAR 20 MPH
13.4 G's
IMPACT
1FT
PROPERLY SUSPENDED RIDER
ROLLS WITH PUNCH
DECELERATION
CRASH FORCES IN THE INTERNAL AUTOMOTIVE ENVIRONMENT ARE INITIATED IN THE EXTERNAL ENVIRONMENT BUT DIFFER FROM THE LATTER.
THE CRITICAL FACTOR INVOLVES THE RELATIVE CHANGES IN VELOCITIES BETWEEN RIDERS AND THEIR COMPARTMENTS.

Fig. 5. Graphic representation of the external and internal automotive crash environments with regard to the effects of decelerative distances on the free and properly suspended rider. The chief difference derives from the fact that this is measured in feet on the outside and fractions of an inch on the inside. The latter explains how a low *g* accident on the outside may be converted into a very high one on the inside.

tive design (DeHaven, Cornell Automotive Crash Injury Research, Crash Injury Research of the Indianan State Police (Sergeant Paul)). A start in this direction has been made, most notably by Ford and Chrysler (Fig. 9) (Table 1).

Forces acting on the rider wearing safety gear (belt) are applied to the body in a predetermined manner to areas which are known to be highly tolerant to linear decelerative forces (hips, abdomen, and chest in the case of shoulder harness). Occasionally, it is to be expected that this kind of force exchange method may result in injury to the rider—usually, however, a small price to pay for protection against more severe effects. Clinicians and radiologists especially should remain alert to this possibility in cases which complain of abdominal discomfort afterwards. Belt designers are fearful that whip-lashing effects may be increased through the use of safety belts (Fig. 10). Thus far, in practice (in addition to the case reported in Figure 10) I have seen only one instance of low back pain from a belt, that of an individual who rolled his car several times in a stock car race without further injury. Roentgenograms of this man's back showed a congenital spondylolisthesis, another illustration of the crash-impact engineering point of view in reverse, *i.e.*, underlying

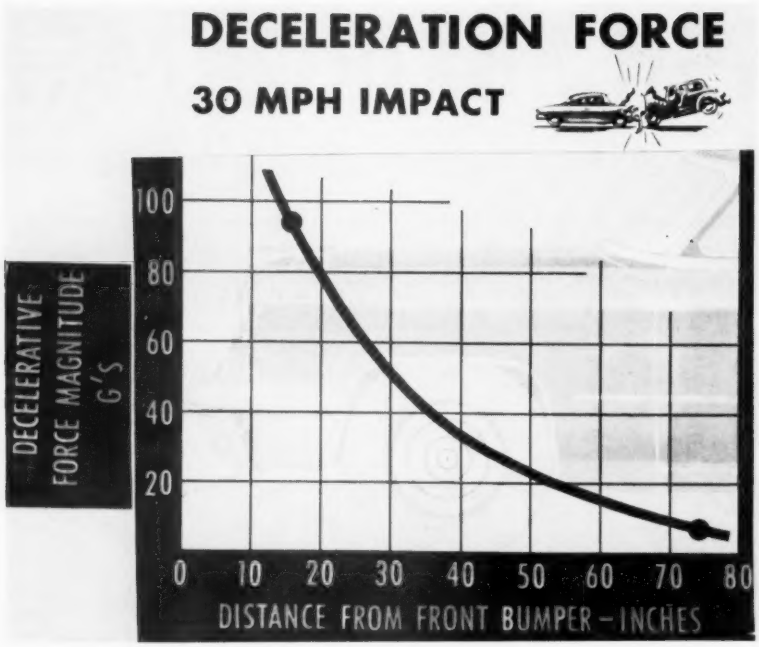


FIG. 6. Magnitude of decelerative force versus decelerative distance. (From A. L. Haynes of the Ford Motor Company.)

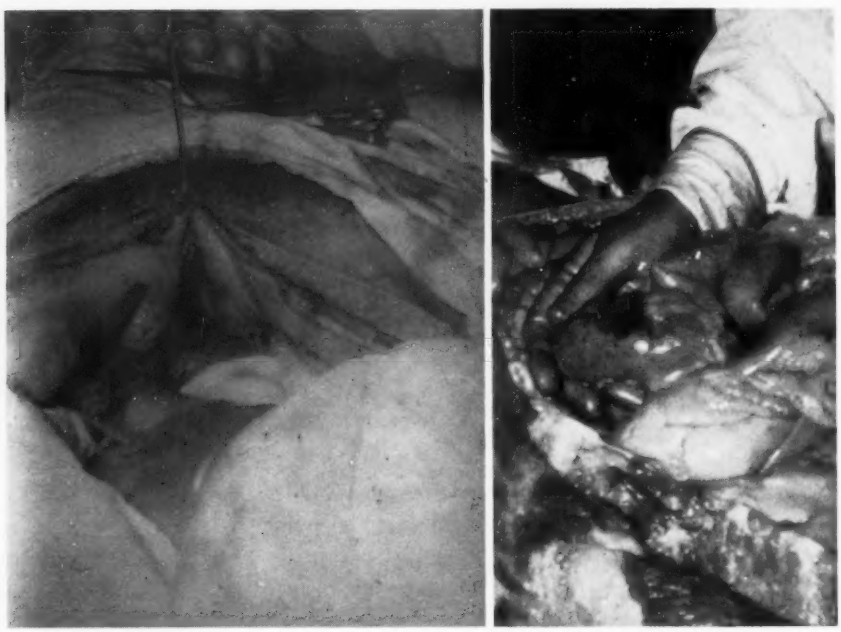


FIG. 7. Photograph at autopsy showing ruptured urinary bladder and contusion of kidney. Always look for the former in victims who were forcibly ejected as this one was. Gross injuries of the urinary bladder rarely occur otherwise.

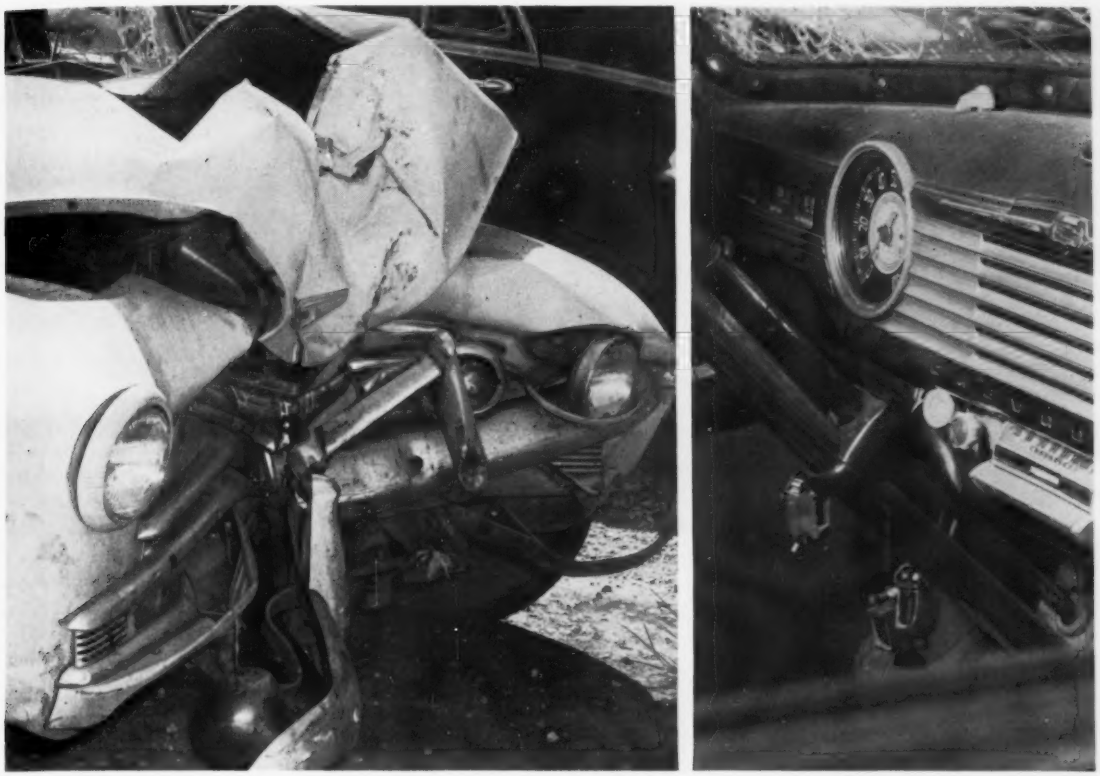


Fig. 8. Decelerative distance on the outside versus decelerative distance on the inside. Man driver fell asleep and rammed a tree at night. The small depression to the right of the deformed steering column was caused by his impacting knee with resultant comminuted fracture of the lower end of the femur. A wide variety of knee-femur-hip-pelvis injuries may result from basic knee-dash impacts.

Table I

PRINCIPLE OF COUNTERBALANCE OR FORCE EXCHANGE: REDUCTION OF INJURIES IN SURVIVABLE ACCIDENTS

Forces Acting on Rider	Pattern of Injury	Moderation of Injury
Impulsive	topical and fractures	energy absorbing surfaces (inbending)
Crushing	internal	stiffen body structure
Inertial	sprains and internal	controlled crumple rate (deformation)*
Ejection—all kinds	frequently internal	safety belts (etc.) safety door locks

* A. J. White of Motor Vehicle Research, taking a cue from the benefits derived from hay bales at critical points of a road race course, has suggested marginal plantings of high energy absorbing shrubbery (Japanese firs) along the highways where accidents are likely to occur because of poor highway engineering or lack of it.

Recommendations for moderating injuries have come from research engineers and researchers in the laboratory and field. I have merely summarized these suggestions and attempted to correlate the kind of injuries which might be especially attenuated through particular types of crash-impact engineering. Physicians should and can suggest forms in automotive engineering safety design.

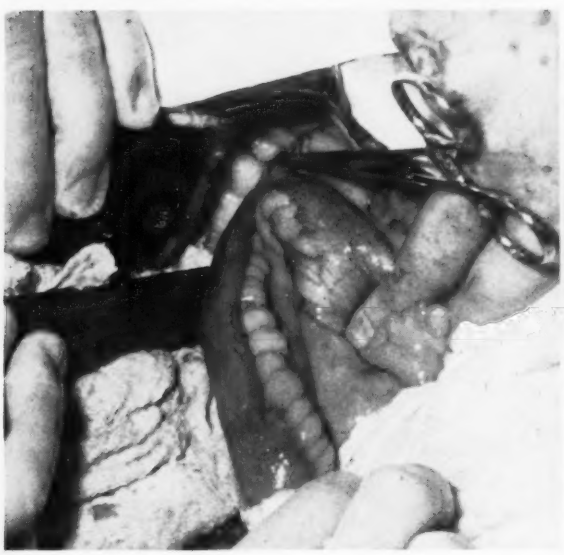
conditions which may lead to greater-than-average injuries under crash conditions.

This entire field, as has already been mentioned, ties in with physical conditions which may also cause accidents. Roentgenographic screening will bring many conditions to light which could lead to so-called pathologic (fractures) injuries (Fig. 11). In particular this recalls osteoporosis of the spine, cystic and other destructive bone lesions, and herniated lumbar disks. The latter sometimes complicates lumbosacral sprains (only rarely so in the cervical region). Occasionally a postoperative disk case becomes involved in an accident, with recurrence of acute symptoms. Myelography is indispensable to the diagnosis in these cases.

PARACLINICAL: FIRST AID

Radiologists can improve first aid by urging against poor handling of simple fractures which sometimes results in compounding. This applies especially to intramural handling of fracture cases to and from the radiologic department. To this end radiologists can also insist upon simpler mobile types of splinting and traction devices. Finally, radiologists should insist upon professional accompaniment of frac-

FIG. 9. Young man driver fell asleep at the wheel and crashed into a bridge abutment. Car was a 1956 Ford. Three aspects of this accident (aside from the one involving the human personality) deserve special mention: (1) direction of impact saved the driver's immediate environment from the collapse of the compartment to his right (to which he owed partially his survival); (2) The so-called "lifeguard" design steering wheel surely held up (deformed only moderately and cracked but not broken in three places), also contributing to his survival, although he did suffer multiple rib fractures on one side; (3) the depression to the right of the undeformed steering column was caused (?) by his impacting right knee, which resulted in a severe dislocation (without fracture) of the right hip posteriorly. The latter was so jammed it required operative reduction.



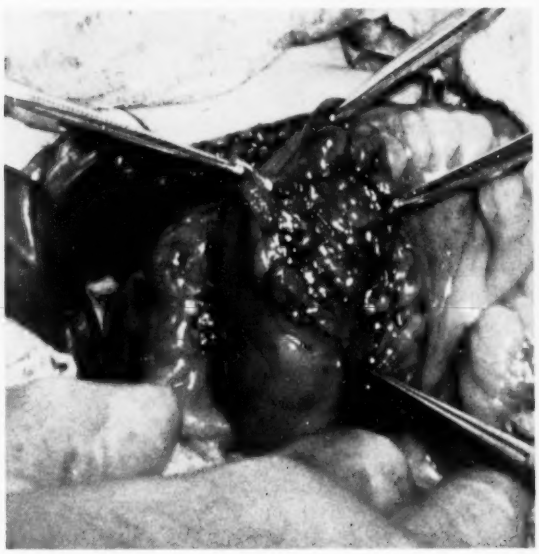


FIG. 10. Chronic adhesion of the terminal ileum to the brim of the right pelvis from the snubbing action of a safety belt worn by the driver in a forward collision with a truck. He braked hard prior to impact and skidded 71 feet (at about 50 miles per hour). Symptoms indicative of partial intestinal obstruction occurred immediately and persisted for several months until they were recognized roentgenographically and confirmed and cured at operation.

Fig. 11. Driver in forward collision who received a compound supracondylar fracture of the femur from a knee-dash impact. However, his knee had fused from a chronic osteomyelitic infection years before. This is another example of conditions prior to accident influencing the effects of crash forces. A stiff hip sometimes transmits excessive compression force to the back and causes vertebral fracture in accidents which do not affect the spines of riders who are otherwise normal.

ture patients whose handling in the radiologic department might cause further injury without the presence of an intern, resident or the attending physician (Table II).

Table II

RULES OF THE ROAD: FIRST AID

1. Remove victims from the wreckage
2. Turn on side for better airway
3. Apply clean dressing
4. Apply tourniquet—only if bleeding is brisk
5. Splint obvious fractures in lower extremity
6. Use sling for fractures in upper extremity
7. Transport safely to nearest doctor or hospital

First aid actually is a preclinical activity, but a most important factor for prophylaxis regarding later care. People will use simpler methods, not complicated instructions. Need is for simplification. Transport should be thought out in some cases. Where specialized types of treatment are indicated (neurosurgery, for example) victims ought to be taken where it is available. Badly injured persons can tolerate transport quite well with effective first aid and in modern ambulances.

CLINICAL

From the standpoint of degree of morbidity motorist casualties which are hospitalized may be classified as follows: 45 per cent are hurt in a more or less minor to moderate degree; 45 per cent are moderately to more severely injured; and 10 per cent are severely to dangerously injured (Fig. 12 and 13). Attention is called to the fact that the mortality rate derives from the latter category almost entirely. This means that any reduction in the mortality of crash victims who reach the hospital must be attained in this subgroup. It is usually this type of patient who needs a team approach to diagnosis and management. Others, for the most part, can be handled successfully by the first physician who is called, provided he has sufficient versatility and experience.

The mortality differentials in motorist casualties should be further clarified and understood. About 15 per cent die outright at the scene of accident. Of the 85 per cent who reach the hospital about two thirds die within the first forty-eight hours, the rest at varying periods of time afterwards. The immediate deaths are due to primary irreversible shock to vital functions. Deaths which occur soon afterwards are also due to primary shock. This is fairly well known and clinically appreciated. Not so well appreciated is the fact that so-called secondary shock is responsible for

FIG. 12. Autopsy pathology within twelve hours after hospital admission: fracture of ribs from impaction of the steering controls, and inertial contusions and hemorrhage of the mesentery, also from the same mechanism. More of the latter may be expected from the wearing of safety belts (?).

most of the deaths later on (Moon). The latter involves considerations of a number of direct and indirect post-traumatic complications (sometimes not distinguished from the primary injuries, a pitfall to be avoided by the alert and astute clinician). These include fat embolism, pulmonary congestive states (and pneumonia), nephrosis, infections, operations and post-operative complications, and even degenerative changes in the body organs.

The rapidity with which secondary shock appears and encroaches upon the factors of safety of the human body are seldom fully recognized and combated to the fullest extent possible. This situation begins with the admission of the victim in the outpatient department of the hospital. This is the place to think about secondary as well as primary shock (Table III).

EMERGENCY CARE

From the standpoint of primary shock, chief attention is focused on the maintenance of the circulation (and arterial volume) and the provision of an adequate airway (Table IV) (Fig. 14). In anticipation of secondary shock states later on, adequate screening of the head, chest and abdomen by roentgenograms should be done (as baseline or diagnostic) in all

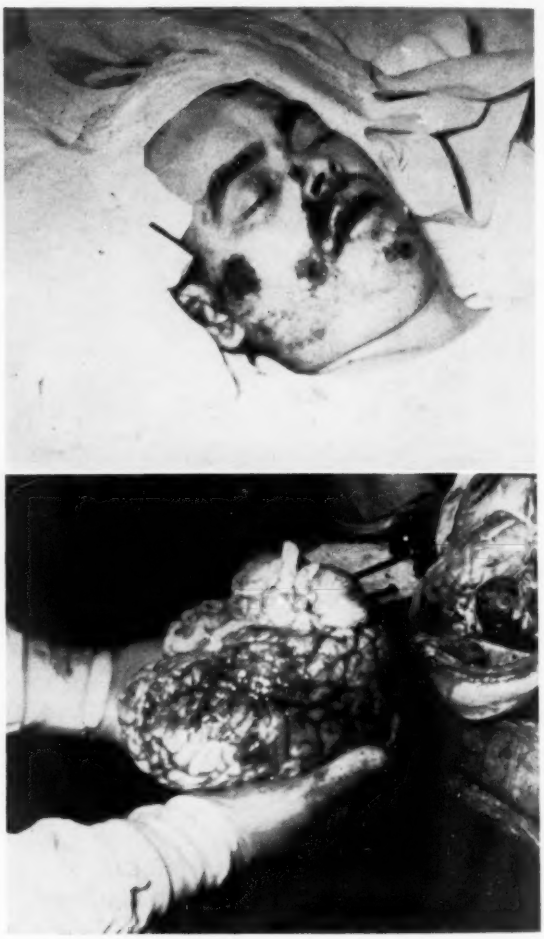


FIG. 13. Autopsy pathology within twelve hours after hospital admission: contusion of the brain with subdural hemorrhage, but without skull fracture. In about 25 per cent of fatalities studied by the writer (series of 30 cases) of the head and chest, approximately 25 per cent of cases had internal injuries without overlying fracture and vice versa.

Table III

BASIS OF EMERGENCY TREATMENT: 19 EARLY DEATHS WHO CAME TO AUTOPSY

Primary Internal Injuries	Primary Shock	Secondary Shock	Total No.
Chest	7	6	13
Head	4	9	13
Abdomen	3	3	6
Massive hemorrhage	5	3	8

Ten cases died of primary shock within a few hours after admission; 9 died of secondary shock within forty-eight hours after admission. Only 1 victim with mediastinal damage died at the scene of the accident; 9 others with varying degrees of mediastinal injury reached the hospital and died as follows: 1 in three hours; 1 in four hours; 1 in eight hours; 1 in nine hours—remainder lived up to eight days after admission. The opportunities for diagnosis and treatment are there

severely and/or dangerously injured persons. This calls for some changes in policy with respect to these patients who are usually considered to be too ill for any extended types of diagnostic ventures. Yet this is the time, or very shortly after admission, to establish baseline data for

Table IV

DICHOTOMY OF EMERGENCY CARE: PROPHYLAXIS AS RELATED TO ULTIMATE MANAGEMENT

(dual responsibility)

Medical Responsibility	Hospital Responsibility
Arrest bleeding	Provide space, tools, personnel
Provide adequate shock	Render first aid
Treat primary shock	Provide blood, plasma and plasma expanders
Sedation?	Provide electrolyte fluids
Check urinary bladder	Provide adequate radiologic facilities
Apply more adequate splints or tractions	Initiate routine laboratory procedures
Determine therapeutic priorities	Observe humanitarian principles during intramural movements of the victim

Complications to remember during admission of the severely injured casualties: primary hemorrhage 66 per cent; all others 34 per cent. The secondary complications (indirect) were infectious 39 per cent, congestive 25 per cent, embolic 16 per cent, nephrotic 6 per cent and degenerative 4 per cent (based on 30 autopsies).

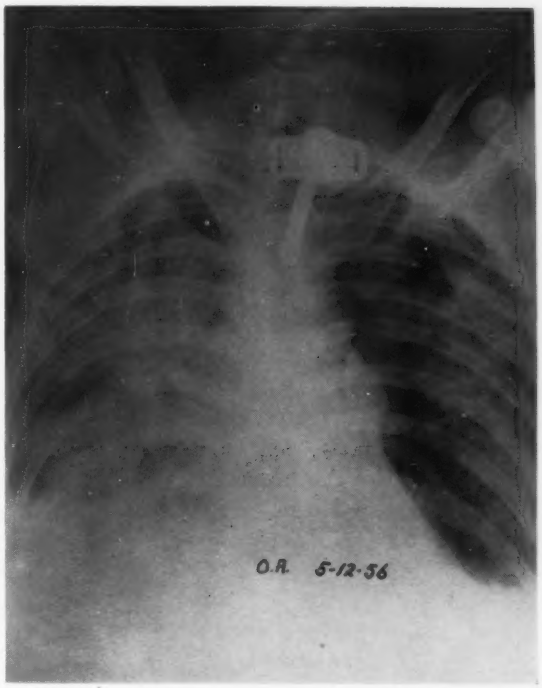


Fig. 14. Roentgenogram of chest prior to death apparently due to a peculiar interstitial collagen reaction in the lungs and glomerular vessels of the kidneys which produced fibrinoid changes in the smaller arterioles of the alveoli and bronchopneumonia and fibrinoid emboli, respectively. The victim also showed an infarction of the pituitary gland (no other brain lesion). On the clinical side he had a fracture of the mandible, crushed chest and a fracture dislocation of the right hip joint (May, 1956). Tracheotomy was helpful, but death occurred on the tenth hospital day without a favorable response to therapy of what appeared to be an ordinary bronchopneumonia. The pathology of mechanical trauma becomes even more complex as it is studied more intensively, especially through autopsy investigations.

the ultimate as well as the immediate care of the dangerously injured individual. Even so the dynamic concept of primary shock is that the patient is getting either better or worse all of the time, but is never in a state of suspended animation. Once primary shock is being treated, there is no point in further procrastination in regard to diagnostic (usually roentgenographic) procedures upon which the patient's life may depend.

There is no rule of thumb here, nor should there be. These risks will have to be

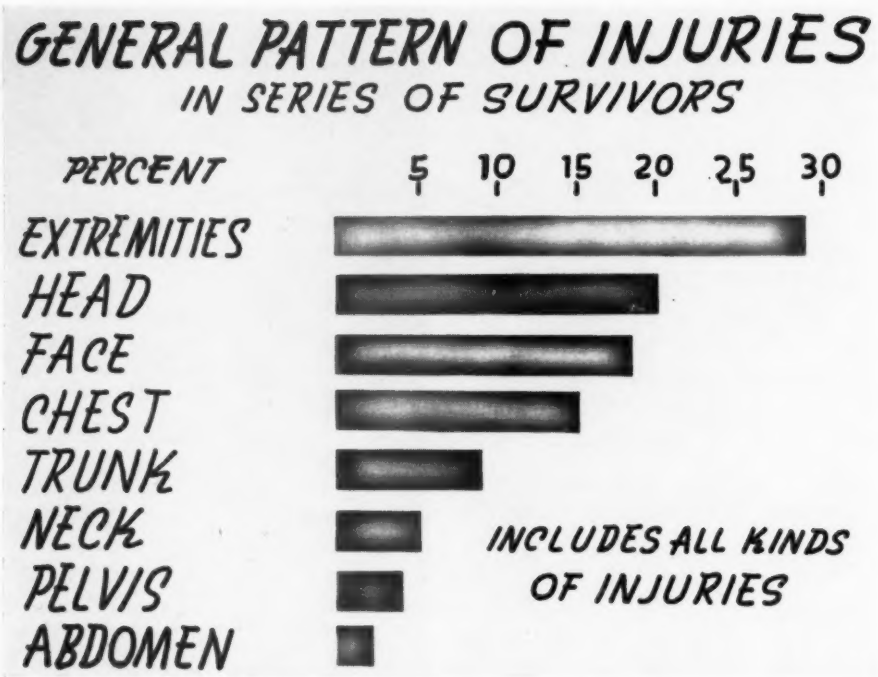


FIG. 15. Rates of distribution of over-all injuries to different parts of the body in a series of over 650 motorist casualty survivors (Missouri Methodist Hospital).

taken—as they are in anesthesia and surgery—and will likewise be minimized with rapid and deft handling of these people early after admission to the hospital. It should be evident that first aid sets the pace and pattern for emergency care. Also, good emergency care can set the pace and pattern of the definitive diagnosis and management of the more severely injured casualties.

DEFINITIVE: ROENTGEN LOAD

This involves considerations of impaired physiology versus medical and surgical management from the standpoint of therapeutic priorities, which cannot be discussed within the scope of this paper. However, an idea of the statistical pattern of injury with regard to the different parts of the body is of special interest and is found to be quite predictable from the standpoint

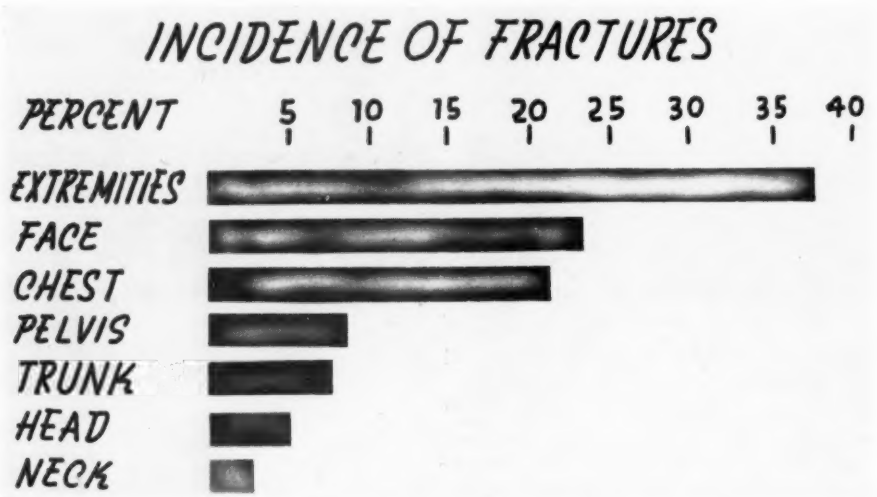


FIG. 16. Rates of distribution of fractures in the same series shown in Figure 15.

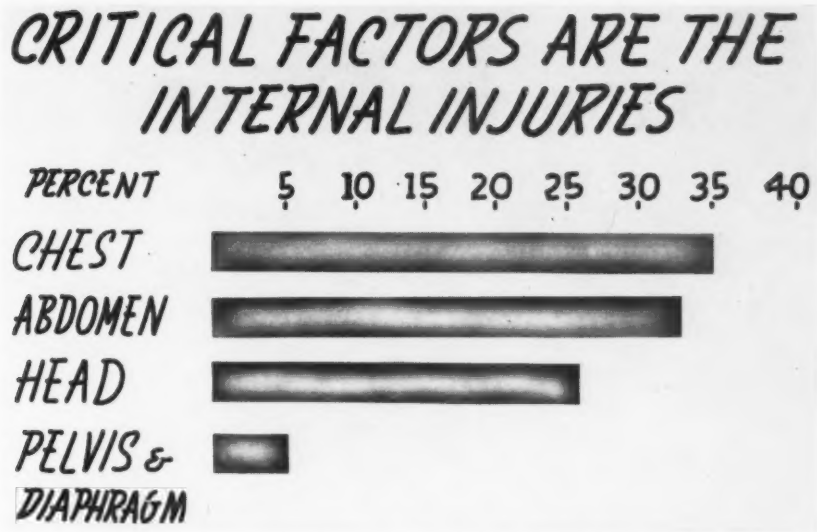


Fig. 17. Rates of incidence of internal injuries in a series of 30 fatalities (Missouri Methodist Hospital, Kansas University Medical Center and St. Joseph's Hospital). The problem of multiple injury (and therapeutic priority) looms ominously from this standpoint.

of over-all lesions (topical, fracture and internal) (Fig. 15). But, from the standpoint of individual patients a great deal of variation is to be expected (for example, with respect to knee-dash impacts). The picture changes somewhat in regard to the rates of fractures to the different parts of the body (Fig. 16). More shuffling of rank results in fatalities in regard to fractures. The fracture rates in a series of 30 fatalities (motorist) were: chest 69 per cent; extremity 50 per cent; head 32 per cent; pelvis 32 per cent and face 14 per cent, respectively (Fig. 17).

SPECIAL COMMENT

The critical factors in fatal cases are the internal injuries. It is becoming increasingly evident that those of the chest are superseding those of the head. This statement is strongly fortified by the fact that in a series of 30 fatalities studied at autopsy, there were 10 cases (14 injuries) which received injuries of the pericardium, heart and great vessels. Despite this frequency at autopsy, such injuries are seldom diagnosed in life or even suspected. In this series this was suspected in at least 2 cases before death. These were not substantiated roentgenologically simply because too much reliance was placed upon portable techniques, instead of regular fluoroscopic ones which would have demonstrated the malfunctioning heart. This was not the fault of the radiologists, but of the clinicians who thought the patients were too sick to be taken to the radiologic department. Radiologists will do well to indoctrinate medical staffs with the difficulties of diagnosis in mediastinal injuries on scout films alone.

POSTCLINICAL: RESIDUAL DISABILITIES

The vast majority of residual complaints from motor vehicle accidents involve the motor-skeletal system, many of which are complicated by medicolegal factors (Fig. 18). These cases require a most careful differential diagnosis (Fig. 19). Most of these cases are seen in office practice, and therefore help to bridge the clinical gap between hospital and later experience.

Analysis of such cases from 1947 to the present from the writer's practice showed a series of 350 cases, two thirds of which complained of neck, low back or combined neck and low back pain. The latter included 91 cervical, 94 low back and 45 combined disabilities. About half were drivers with the remainder divided between

Fig. 18. Residual motor-skeletal disabilities and their medicolegal complications. Note the large number of spinal conditions, also the high rate of medicolegal considerations in this particular series.

those in the front and back seats (about one third in the latter area). Accident configuration was most constant for the cervical group, as has already been mentioned. Otherwise there were all kinds of impacts responsible for the alleged personal injuries.

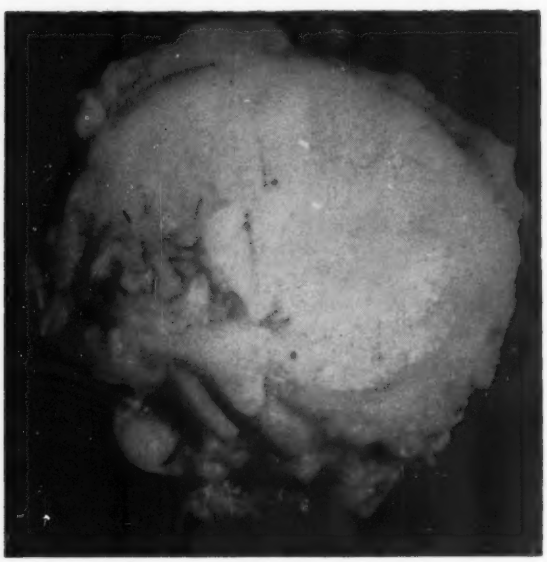


Fig. 19. Patellar articular surface after surgical excision for chondromalacia (and hypertrophic changes as well). Resembles a femoral head with malum coxae senilis. Patellar changes followed a knee dash impact of long standing. Clinical and medicolegal implications are obvious.

Concluding Opinions

Medical concern is growing in regard to the increasing toll of life and limb being exacted daily on our highways. With the advent of epidemiologic techniques and methods of studying these accidents, the evolution of medical participation may be expressed as advancing from a "do-it-yourself" attitude to a "do-it-together" plan of medical action (Fig. 20). The role of the radiologist, henceforth, will become more specific than it already is, both in the solitary and team approaches to problems of medicine. His responsibilities increase with better knowledge of the living morbidity and autopsy pathology of motorist casualties because increasing knowledge impresses clinical rather than surgical challenges alone, as has been thought to be the case in the past. This means alertness toward therapeutic conservatism as well as toward positive surgical intervention which points up the necessity for more participation by internists in the severely

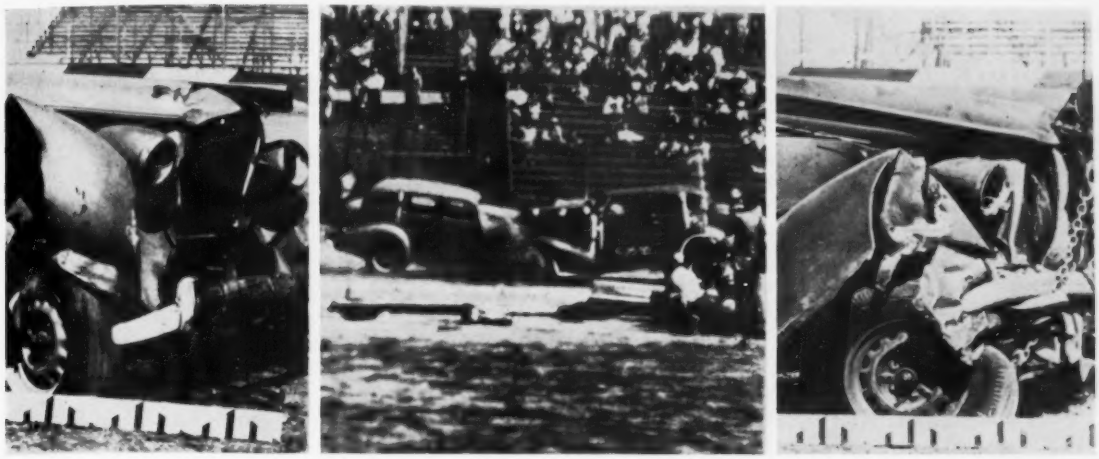


Fig. 20. Symbol of controllableness of accidents and injuries: historic head-on collisions staged experimentally at the University of California (Roth). Highspeed movie photograph supplied by Severy and Mathewson. The fact that motorist accidents and injuries can be studied under controlled conditions of experimentation ("situation injuries") bids well for their ultimate control under traffic conditions.

injured motorists. There is no intent to subordinate the surgical aspects of this problem by this statement. On the contrary it should be a guide toward ever greater accuracy in diagnosis and therapeutic priorities.

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ROENTGEN SCREENING OF MAMMARY TUMOR PROGRESSION*

By J. GERSHON-COHEN, M.D., *and* HELEN INGLEBY, M.D.

ROENTGENOGRAPHY offers a means of surveying the human breast. During the past five years, we have improved criteria for the differential diagnosis of the various benign dysplasias and carcinomas.[16–20,23,34] Small carcinomas may be detected on the roentgen film even in dysplastic clinically lumpy breasts (Fig. 1). A number of occult cancers have been localized and diagnosed.[22] Some of these tumors had already metastasized even though not palpable clinically. A few were less than 5 mm. in diameter on section.

Serial periodic roentgen studies are an eminently suitable substitute for the serial biopsies used by experimentalists in the study of tumor progression; but, could the radiologist equal this accuracy in the discovery of a lesion while still localized and noninvasive? And more speculatively, granted that a breast showing excessive reaction to hormonal stimuli is potentially malignant, could this breast be identified on the roentgenogram and kept under controlled observation?

Most malignant lesions present pathognomonic roentgen signs. In addition to spiculated invasive borders, punctate calcifications are seen on the roentgenogram in approximately 30 per cent of all breast cancers. When numerous and present along the line of the ducts, these deposits are pathognomonic of intraductal (comedo) carcinomas.[21] A less conspicuous pattern of calcification may be found in cases of intraductal carcinoma "*in situ*" (Fig. 2).

Recently we found evidence that certain malignant lesions may be associated with characteristic structural patterns in the human breast. These patterns have been correlated to some extent with those dysplasias that are, to the experimentalist, the expression of hormone imbalance, and hence possible forerunners of malignancy.

Few would forego today the benefits of periodic roentgen examinations of the chest for early detection of pulmonary tuberculosis. Advances in the diagnosis and treatment of pulmonary disease took place after the roentgenologist became proficient in the interpretation of films and later applied survey methods for the detection of early lesions. Likewise, long term periodic roentgen studies of asymptomatic women should establish roentgenographic criteria of precancerous changes in the breast and so lead to more timely surgical measures in the control of mammary cancer. Further advances in treatment of mammary cancer may also be expected with clinical recognition and application of the experimental work on tumor progression.

Clinical studies of mammary cancer pathogenesis in the human are few and incomplete because of the need for extended observation. Recently Kiaer recorded the results of exhaustive long-range observations and reviewed the literature in an excellent monograph. His conclusions point to a relationship between severe fibroadenomatosis and malignant neoplasia in women and confirm strongly the experimental concepts of tumor progression.[35] But the better to realize the necessity of understanding tumor progression in its relationship to histologic and roentgenographic observations, resort must be made to the findings of experimental tumor progression.

EXPERIMENTAL TUMOR PROGRESSION

Literature on experimental tumor progression is derived from two sources: (a) clinical and pathologic observations of spontaneous tumors in experimental animals, and (b) study of tumors induced experimentally by chemicals, viruses or hor-

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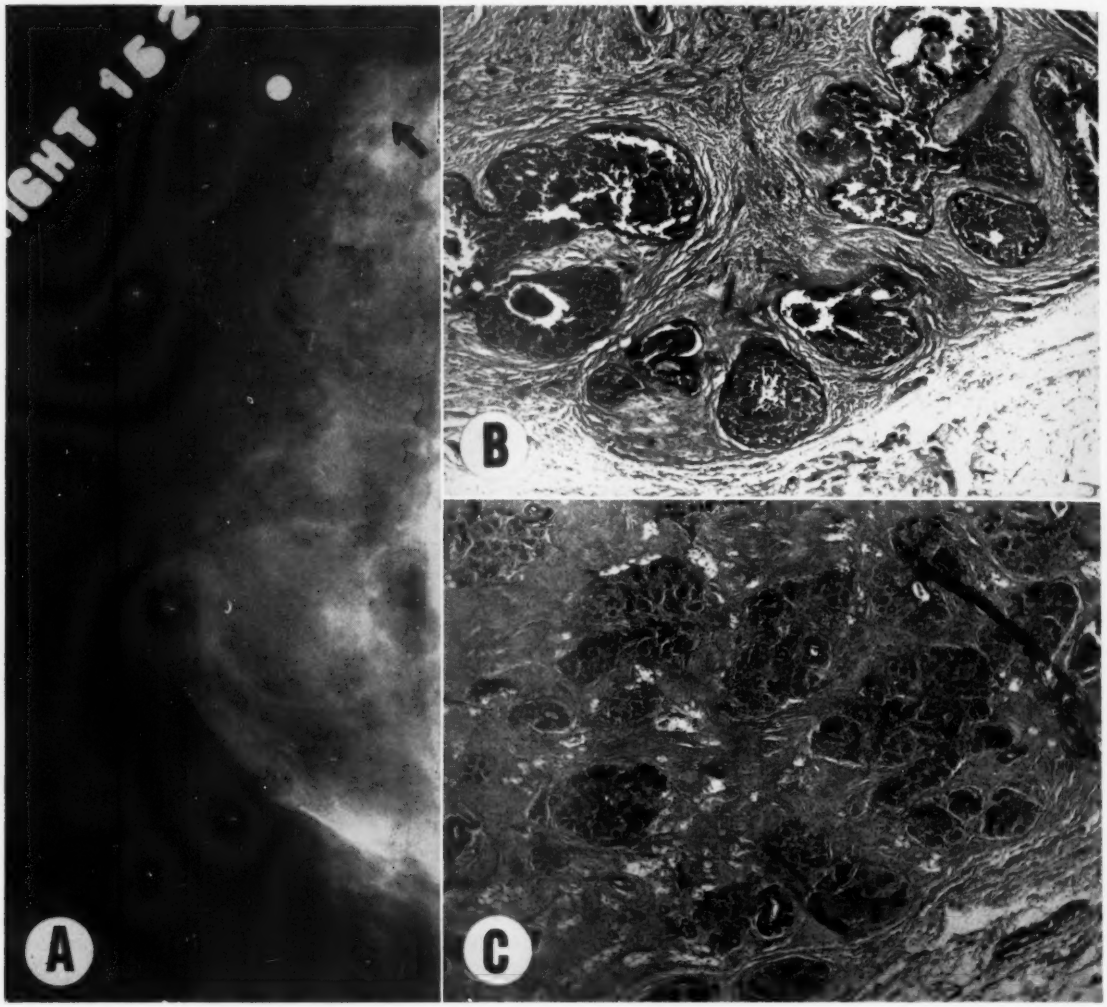

FIG. 1. Duct carcinoma—adenosis. C 1520, aged forty years. Multiple nodules in both breasts. One rather firm freely movable pea-sized mass in the upper outer quadrant of the right breast. (*A*) Film shows multiple opacities characteristic of adenosis. Directly under the lead marker on the skin is an ill-defined opacity exhibiting punctate calcification. Diagnosis: duct carcinoma, adenosis. Histopathology: (*B*) Duct carcinoma, 1 cm. in diameter (×35). (*C*) Adenosis (×35).

mones. An excellent review of the subject, actually a new look at tumor pathogenesis, is the one by Foulds.[8]

Spontaneous Tumors. Greene described mammary neoplasms in rabbits, originating most commonly in breasts with preexisting cystic changes similar to Schimmelbusch disease in women, or less often as adenomas in apparently normal breasts. In the former case the disease grades from cystic disease, through noninvasive neoplasia to cancer with metastases.[26–29] Greene found similar progressive changes in uterine tumors of rabbits, verified by repeated biopsies and transplantations.[30]

Foulds studied tumor progression in a hybrid strain of mice and their inbred descendants.[9] The majority of mice had multiple mammary tumors before death. Some of the tumors grew progressively from their initial appearance to death of their host. Others were hormone responsive and grew only during pregnancy; following parturition they regressed partially or completely. There was no apparent ratio or order of occurrence for the two tumor types

in individual mice.[10] Hormone responsiveness in a tumor persisted sometimes through repeated pregnancies, covering most of the animal's life-span. However, all adequately studied tumors lost hormonal responsiveness permanently after a time. In some instances a tumor remained hormone responsive during the lifetime of the primary host; but if the life of the tumor was prolonged by transplantation, reversibility was eventually lost and progression to malignancy inevitably occurred. Rate of growth and hormonal responsiveness were independent of each other in individual tumors. Where multiple tumors were present, they progressed one by one. Abrupt change from a responsive to an unresponsive hormonal state was the most conspicuous and frequent manifestation of malignant progression.

A distinctive histologic feature of Foulds material was early carcinoma-like changes in the proliferative and sclerotic plaques where hormonal sequences could still be traced.

Experimental Tumors. In the study of tumor progression causative factors are of little consequence. Whether carcinogenesis

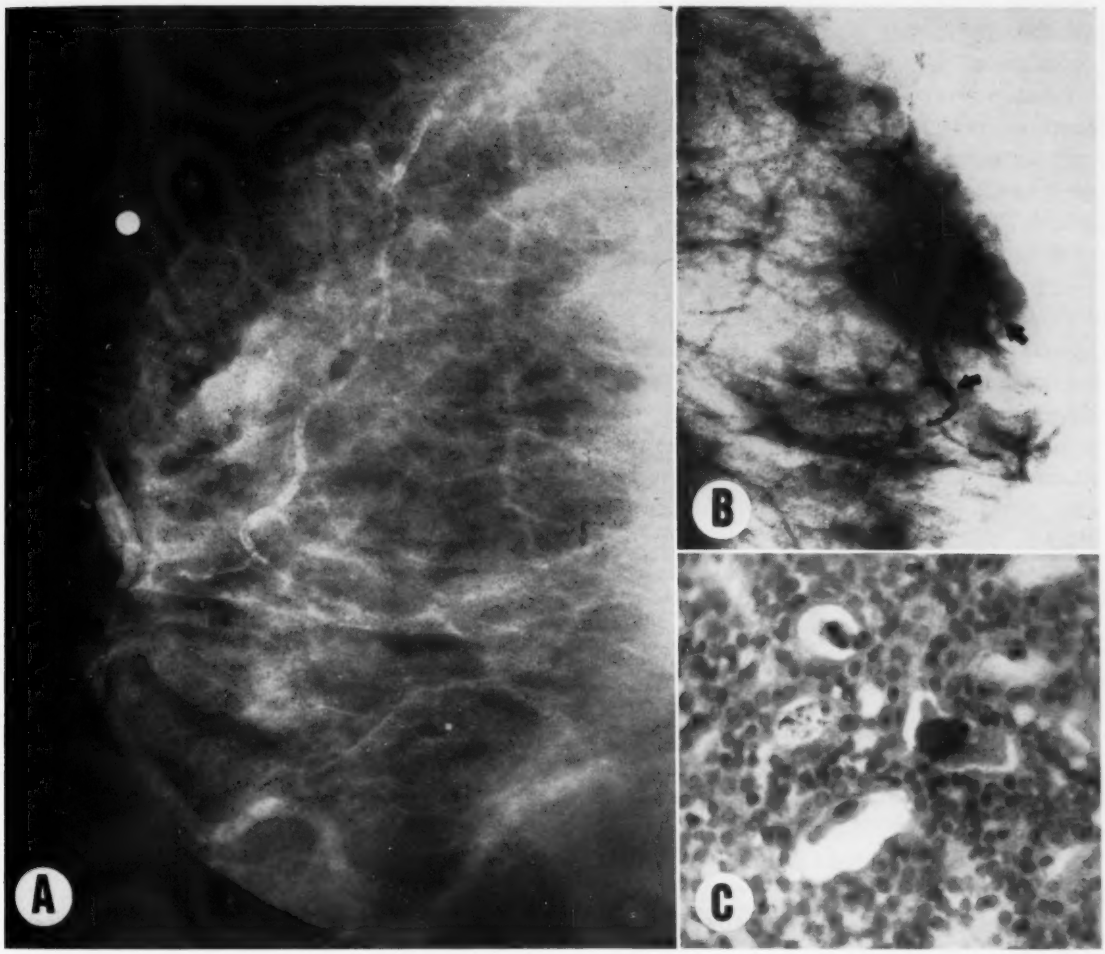

Fig. 2. Carcinoma *in situ*. C 1013, aged seventy-three years. Serous discharge from left nipple of two months' duration, bloody at times. No palpable masses. (*A*) Atrophic breast showing irregularly dilated ducts chiefly above the nipple. Nipple slightly retracted. A calcified vessel crosses the breast. (*B*) Hyper-contrast reversed print to demonstrate calcification. The upper arrow points to a cluster of nonpolarized calcific deposits, the lower arrow to the calcified artery. Diagnosis: intraductal hyperplasia, possibly malignant. Histopathology: (*C*) Carcinoma *in situ*. Commencing calcification of necrotic material (×480).

is caused by chemicals, hormones or viruses, progression to malignancy follows the same pattern.

Rous and Kidd applied tar to the skin of rabbits. The warts which first appeared required continuous stimulation for growth.[44,45] Despite continued tarring few papillomas developed into carcinomas and only after long delay. Early workers were misled into a too frequent diagnosis of cancer; the "factitious malignancy" of carcinomatoids, despite histologic signs of invasive growth, regressed unless external stimulation was continued. Tumors that regressed in intermissions of tarring recurred at the same site with the same specific characters when tarring was resumed. However, nonspecific irritants (such as turpentine, which by itself induces no tumors on normal skin) could evoke growths from previously tarred areas at the site of tumors which had regressed. Alternating regression and recurrence could be repeated two or three times at one site.[37]

The papillomas induced by Shope papilloma virus also confirm the theory of tumor regression. Some Shope papillomas tend to malignancy from the first by a continuous series of alterations. Others, benign at first, change into malignant papillomas and finally into squamous carcinomas.[43] Pathogenic progression of these papillomas to the final malignant phase of growth can be hastened by trauma, infections or chemicals such as scharlach R.

Directly pertinent to the problem of human mammary cancer progression are experimental studies of tumors provoked by hormonal imbalances, although genetic factors may be considered an additional influence.[1,2,12–15,39,42] The pituitary, testis and thyroid of rodents may display neoplasms, the growth of which is sustained only in an unbalanced hormonal environment. These tumors are transplantable only to animals with the same hormone imbalance as the primary host. Many of the growths do not advance beyond the adenoma stage in the life-span of the primary host. But tumor progression takes place in serial transplantations, eventuating in carcinomas which then grow independently of unbalanced hormonal stimulation. Hormone-induced hyperplasias may advance through benign neoplasia to malignant neoplasia without abrupt or distinct cleavage between successive stages.[4,36] Even more important to the clinician is the experimental observation that estrogen-provoked tumors of the breast and uterine cervix in rats and mice may develop long after withdrawal of the hormone, and after the initial hyperplasia has waned. There is also evidence of discontinuous pathogenic progression through successive stages in which hormones are not the sole etiologic factor.[7,14,24,41] Progression from adenoma to carcinoma in the thyroid of rats may be provoked by goitrogens like thiourea.[32,33]

THE HISTOLOGIC DIAGNOSIS OF TUMORS

Confronted by hormone-responsive tumors, the histologist has no absolute criteria by which to spot lesions destined to become malignant. Behavioral characteristics are not necessarily related to those which determine histologic appearance.

Four behavioral qualities characterize a tumor. They are: (1) invasion, (2) dissemination, (3) rate of growth, (4) responsiveness to hormonal stimulation.

Glücksmann showed that the rate of cell proliferation and the rate of individual cell growth in the mouse are not necessarily different in malignant tumors and benign warts.[25] Bonser showed that some testicular tumors induced by her in mice are histologically malignant, but lack the power to grow without estrogenic stimulation.[2] Some transplanted thyroid tumors in mice lose responsiveness to hormonal stimulation without change of histologic structure.[40] Spontaneous uterine tumors in rabbits, carcinomatoids of rabbits' skin and hormone dependent tumors in general show little relation between histologically observed malignancy and their capacity for independent growth. Greenstein has shown that the enzymatic pattern in transplanted tumors of like origin is independent of

growth rate.[31] These experimental observations already have alerted leading histologists to take a new look at human biopsy material in formulating a diagnosis of malignancy.

Foulds previously emphasized that it is insufficient to label a tumor malignant; it is also necessary to define its characteristics and to what degree it is malignant.[8] Classical text-book descriptions of tumors, even though based on associated clinical and pathologic observations are inadequate because they fail to consider all qualities of tumor behavior.

Inconsistent tumor behavior is exemplified by locally "malignant" tumors which are highly invasive but do not disseminate, by metastasizing "benign" tumors which disseminate without notable local invasion, and finally by undoubtedly malignant tumors that are responsive nonetheless to hormonal influence.

Chemotherapy often can control a growth for long periods of time, but a shortened terminal phase frequently counterbalances the phase of remission.[3] Clinicians have already learned this from hormonal management of human breast and prostatic cancer. The independence of tumor growth-progression to hormonal responsiveness accounts for the frequent discrepancies in clinical evaluation of a tumor based merely on its size. A small tumor may be far advanced in growth-progression. Conversely, a large tumor may be at an early stage of growth-progression, and its size and duration may be due merely to long and/or vigorous hormonal stimulation.

Each of the four qualities accounting for tumor behavior has different intensities and end-points. Moreover, some tumors often regress completely and permanently. Even malignant tumors may regress.[11] The mammary tumors of mice observed by Foulds[10] revealed wide and independent variations in grade of tumor progression. These variations in tumor progression are mirrored nicely in the roentgenographic patterns of the dysplasias and neoplasias of the human breast.[17,18,19,34]

THE PROBLEMS OF EARLY DIAGNOSIS

Even if a definitive cure for cancer were to be discovered tomorrow, early diagnosis would still remain of paramount importance. In this respect, cancer is comparable to diseases such as tuberculosis, where the initial lesions engraft themselves with little or no manifest reactions. Yet for best results, treatment is most effective when begun in the pre-clinical stage.

In the human breast, puncture of a cyst with removal of fluid, removal of an adenoma or biopsy of a hyperplastic lesion now are done for lack of more effective control measures. Merely to get a biopsy of a tumor which has become clinically manifest is to gain no insight into other parts of the same or opposite breast. This is especially important, since in both spontaneous and chemically induced tumors, only one of multiple tumors usually progresses to malignancy.[5,6,46,47] There is no better way of keeping sight of all tumors in a breast than with a good roentgenogram.

Clinicians of wide experience agree that cancers are usually far advanced in growth-progression by the time a woman reports to her physician with a lump in her breast.[38] An over-all five year survival rate of only 27 per cent in spite of improvements in radical surgical and irradiation techniques testifies to our helplessness in the face of established breast cancer. The chief hope today lies in the detection of the earliest stages of cancer and of the precancerous states. We believe that the best approach to this problem of early diagnosis is by means of roentgenography and the judicious use of survey techniques.

Difficult as it may be to acquire skill in the roentgen interpretation of mammary disease, the roentgenologist must train himself to ferret out breast lesions before growth has advanced beyond the primary focus. The art of interpretation is best learned by studying films of breasts in women admitted for biopsies or amputations and comparing them with the surgical specimens. Each radiologist must do this for himself since the reading of reports and

texts is no substitute for the acquisition of experience. Many radiologists who have given mammary roentgenography "a fling" complain that the number of mistaken diagnoses is too great. What they fail to realize is that when they first learned to interpret films of the chest or gastrointestinal tract or any other system, they likewise made numerous mistakes. Therefore, to emphasize mistakes due to inexperience at the expense of the higher percentage of accuracy that comes with adequate training is obviously not the way to make progress in this field of endeavor.

CONCLUSION

Surgeons and histologists have no means of following tumor progression except possibly by multiple serial biopsies. Since this is impractical in the human, the use of periodic roentgen examinations of breasts in women over thirty-five years of age is suggested. Especially is this stressed in women who have had a breast operation for a benign lesion.

It will take time for the roentgenologist to become familiar with the appearance of abnormal breast changes so that he can recognize the earliest patterns of premalignant processes. It will require courage for the surgeon to take action when classical clinical signs of malignancy are still absent. It will call for insight from the pathologist to appraise the vagaries of tumor progression. But with joint cooperation, improvement in survival statistics may be achieved by radically changing the timing rather than the extent of surgical interference.

A clinic for the periodic roentgenographic survey of the breasts of healthy women over thirty-five years of age has been inaugurated at the Albert Einstein Medical Center, Northern Division.*

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* This project will be aided by a grant obtained through Dr. Joseph C. Doane, recently Medical Director and Chief of Medicine, Jewish Hospital, now the Northern Division of the Albert Einstein Medical Center, Philadelphia, Pennsylvania.

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PERMISSIBLE DOSE PROBLEMS WITH SPECIAL REFERENCE TO DIAGNOSTIC PROCEDURES

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IT IS the purpose of this paper to discuss permissible dose problems with special reference to diagnostic procedures and to describe a method for recording radiation dosage resulting from these procedures.

In a previous paper[13] a plea was entered for the adoption of a personal radiation diary in order to reduce to a readied exactness the composite previous radiation record of any individual patient and to avoid supratolerable levels.

Before any personal diary for the patient's permanent possession can be elaborated, it is necessary to appraise the present methods of recording roentgen radiation exposures.

This appraisal quickly yields the following information: (1) that all roentgen radiation administered for therapeutic purposes by Diplomates of the American Board of Radiology is now nationally and very satisfactorily recorded in the respective departmental records on forms which have been standardized and which are all almost alike (no copy of these records is given to the patient); (2) that at present there is no known routine recording of radiation dosage received from purely diagnostic procedures in any general hospital.

Accordingly, the recording problem becomes limited to the proper recording of such exposure administered during diagnostic procedures either by fluoroscopy, roentgenography or both.

Inasmuch as our previous paper[13] had also tabulated the doses delivered to an adult patient during fluoroscopy and roentgenography, the immediate problem resolved itself into finding a simple recording method which could be used routinely without additional expense in such a way as to be permanent, presentable, readily available and easily applicable and understood.

According to Failla,[6] the requirements of clinical radiation dosimetry are becoming more stringent, compelling the radiologist and others to become more thoroughly familiar with the fundamentals of the dosage problem.

Recent discussions have pointed up the problem of radiation dosages delivered during diagnostic procedures and their increasing importance, especially so since we now appear to be in a period during which the permissible dosage is being reappraised and possibly graded downward from the present 0.3 r per week. For example:

1. Hansen and Madsen[8] favor a reduction in the tolerance dose to 100 mr (0.1 r) per week.

2. Muller[12] states that the so-called daily tolerance dose of 0.05 r per day or 0.3 r per week when applied persistently to mature animals for the remainder of their lives could in a man, for example, shorten his life by one and one-half years.

3. Brucer[2] states that the maximum permissible dose level of exposure (300 mr total body exposure per week) is not a "safe level." Rather, the least possible exposure is the safest and the 300 mr level is a maximum.

4. Ernst[5] makes reference to the confusion regarding the roentgen tolerance dose in various countries in relation to the amount and the time factor. As for example, the tolerance dose in Germany is 0.25 r per day, in Sweden it is expected soon to be 0.1 r per week, in the United States and Canada it is 0.3 r per week, and in England 0.5 r per week. Ernst proposes that the roentgen tolerance dose should be subdivided into various categories, the most important of which, as applied to the scope of this paper, would be the permissible average dose rate which represents the permissible average dose per unit of time. He suggests the following table:

	Hard Radiation		Soft Radiation	
Life	200	r	500	r
Year	6	r/yr.	15	r/yr.
Week	0.15	r/wk.	0.35	r/wk.
Day	0.03	r/da.	0.65	r/da.
Hour	0.006	r/hr.	0.025	r/hr.
Minute	0.0001	r/min.	0.0004	r/min.

Batho[1] defines the tolerance dose of radiation as that dose below which level the natural processes can replace completely the damage sustained, but he and Muller stress that there is no tolerance dose for genetic injury.

According to Failla,[6] as the field of medical radiology expands, the dosimetry problem becomes more complex. The International Commission on Radiological Units has taken the initiative and assumed responsibility for promoting the compilation and distribution of information relating to units and dosimetry on a continuous basis rather than at intervals coinciding with the International Congress of Radiology.

The reason for differing opinions concerning the "tolerance dose" is that neither the criteria nor the methods for measuring radiation damage are well defined. Animal and human experiments are not necessarily comparable, especially with respect to genetic studies.

In recent years there has been a corresponding concern over the skin doses received by the patient during routine diagnostic studies. A simple method whereby all skin dosage during various area examinations may be quickly computed was devised by Sorrentino and Yalow.[15]

The patient may be unwittingly overirradiated because of poor judgment on the part of the radiologist or may suffer from the cumulative effect of successive examinations when a full history of earlier exposure has been withheld or disregarded. Quimby,[14] in discussing patients who, for diagnostic purposes, are subjected to such a degree of radiation that acute or chronic changes are produced, stated that with suitable calibration of equipment and with an understanding of the dangers on the part of those using it, this problem should disappear. This is particularly true if an educational campaign is conducted to alert nonradiologists, who use fluoroscopes to look for foreign bodies, to set fractures, and to conduct heart and lung examinations. We believe that this educational program should include proper recording of all forms of radiation in a personal radiation diary.

Daland,[3] in discussing damage to the skin from unwarranted and unexplained radiation, cites several types which make up this group. Among these are untrained and/or careless physicians using irradiation, patients injured by excessive fluoroscopy, damage from overirradiation of growing epiphyses, psoriasis, plantar warts and fungus diseases, and damage from overirradiation when a depilatory effect is sought. He stresses avoidance of irradiation a second time and warns against patients applying to various physicians for further treatment without transfer of proper records.

Handloser and Love[7] state that the average person experiences no significant hazard from roentgen examinations but that in workers handling radioactive materials such roentgenographic exposures may produce an excessive cumulative effect. If a person is exposed to radiation in the course of his work, it is desirable to know the radiation doses resulting from common diagnostic roentgenologic procedures even though these do not represent total body irradiation.

This compounding of effects may also be developing in wards in which many patients receive different radioisotopes in relatively large quantities. Consideration of this exposure hazard will affect the design of radioiosotope wards (Brucer[2]). Protection must be afforded for the patients from each other and for the personnel who make even a short term career of handling these patients. At the Oak Ridge Institute of Nuclear Studies,[2] the quantity of exposure is measured by film badges for secretaries, visitors and janitors, while those actually exposed, such as nurses, technicians and

staff members, wear pocket chambers which are read twice weekly by a health physicist and a continuous record thereof is kept. Selected personnel in close contact with large radiation sources or those who may be expected to have the greatest exposure wear a direct reading dosimeter. In special circumstances, palm and finger badges, wrist and forehead chambers and other monitoring devices supplement these standard procedures.

Macht[9] is now conducting a study to determine the possible effects of ionizing radiation on the genetic structure of man in order to determine whether the present tolerance limits and protective standards are satisfactory. This is to be accomplished by a questionnaire sent to about 4,000 radiologists and other radiation workers and to an equal number of physicians not exposed to radiation.

The significant incidence of leukemia in radiologists compared with the nonradiologic physician population is now well documented. March[10] has shown that leukemia is over nine times as great in radiologists as in nonradiologic physicians and is twice as great in all physicians as in the general adult white population. Most of the increased incidence of leukemia in physicians as a whole is accounted for by the inclusion of radiologists in these statistical data. However, even with the exclusion of radiologists, there is still a slightly higher incidence of leukemia in physicians when compared with the general adult white population. This can be accounted for by the rather considerable number of nonradiologic physicians who use fluoroscopes, diagnostic roentgenologic units, radioactive isotope material and radium in their work, frequently with little or no adequate knowledge of the dangers involved and resultant inadequate protection. This points up the importance of widespread education of all physicians who use roentgen radiation and particularly brings into sharp focus the need for an accurate method of recording dosage in roentgen radiation exposures for all individuals.

The present method consists of stamping the roentgenographic envelope as shown in Figure 1. This will shortly be replaced by printing the same figure on the original roentgenographic envelope.

The clerk in charge of setting up the envelope enters the date and the type of examination in the appropriate boxes. The remaining boxes are filled in by the radiologist after he finishes the interpretation. If our previous paper[13] is consulted it would be readily appreciated that this is a very simple and almost automatic procedure. In instances where fluoroscopy has been employed, the exposure time obtained from a timer is translated into its roentgen equivalent utilizing the proper line on the table as determined by the kv., ma. and target skin distance for the particular installation. The entire computation and recording procedure requires only a very short time.

In this manner it is possible to state very quickly and accurately what the patient has received, to what area and when. At a later date when a personal radiation diary is finally adopted, it will become a simple matter to record therein exactly what the patient had received.

In order to simplify the recording procedure, we have considered the entire dose to be delivered to a single area of the body. Since, in general, it is difficult to delineate each area of exposure exactly, it was felt wisest to be conservative and determine the maximum dose resulting from any procedure.

No small part of the confusion in setting the limits for a tolerance dose lies in determining the biologic factors which are to be considered in evaluating radiation injury as well as in estimating the radiation received by the injured part. It would, therefore, be of some value to consider the volume dose received during roentgenographic examinations compared to the 21×10^3 gram roentgens received in one week of generalized body radiation by an average 70 kg. man exposed to the accepted tolerance dose of 0.3 r in one week.

FIG. 1

According to Mayneord,[11] the volume dose can be approximated using the relationship:

$$\text{Vol. dose} = 1.44\ T_{\frac{1}{2}}^{1} AS$$

where $T_{\frac{1}{2}}^{1} = \frac{1}{2}$ value thickness in tissue for the radiation

A = area irradiated
S = 1.2 × dose in air at the skin.

For a typical gastrointestinal series, the area irradiated in the average single film is 10 × 12 cm.^{2}, $T_{\frac{1}{2}}^{1}$ is about 4 cm., and the dose in air at the skin is about 30 r. The estimated volume dose, using these assumptions, is then 25×10^3 gram roentgens. Thus, the volume dose in both instances is approximately the same.

Since the problem of radiation dosage to which the radiologist is exposed in his everyday work is of equal importance, a suitable method of recording this exposure is fundamental. Accordingly, we have initiated a system which records with reasonable accuracy the amount of radiation exposure obtained during fluoroscopic procedures by each individual physician, resident, full time and consulting staff. At

least once, and preferably twice yearly, the radiation received by each member of the radiologic staff who performs fluoroscopy is measured for an entire morning's fluoroscopic examinations, in a number of positions, using pocket dosimeters. These dosimeters are distributed on the forehead, forearm, underneath the lead apron over the waist, in front of the lead apron over the area of the waist, ankle, middle of the back and inside one of the lead rubber gloves. These dosimeters are attached in place in each area with adhesive and a direct reading in milliroentgens is obtained at the end of the fluoroscopic procedures. These readings are recorded in their proper column (See Table 1). The dosage received by each member of the radiologic staff is then obtained by multiplying the number of daily periods spent in fluoroscopy during the year by the average dosage received in one period. This is, of course, a gross method, but we feel that it is useful in evaluating the radiation dosage received during fluoroscopy in the course of a year without the need for using a multiplicity of film badges especially calibrated for the quality of roentgen radiation employed in diagnostic roentgenology. Keleket dosimeters are used for these surveys. A calibration chart supplied with these dosimeters indicates that in the voltage range used in diagnostic roentgenology the reading obtained is high by a factor of about 1.7. This correction can be made if desired.

Monitoring is not required for personnel working outside radiologic rooms in either the diagnostic or therapeutic radiology sections. All rooms in these sections have been surveyed and the radiation levels outside these rooms is such that personnel would not receive a detectable amount of exposure in a reasonable time.

The problems involved in detecting and recording of radiation exposure of personnel either working with radium or artificial radioisotopes or coming in contact with patients who contain hazardous amounts of such radioactive material will be considered separately at a later date.

TABLE I

RADIOLOGY DEPT. STAFF FLUOROSCOPIC EXPOSURE JAN. 21–26, 1954

Name and Date	Dosimeter Number	Position	Milliroentgen Reading Before	After	Dx	Fluoroscopic Time	Type of Examination
Dr. J. Doe	1	forehead	30	31	1	14 min.	2 G. I.
1-21-54	2	r. shoulder	5	5	0		4 B. E.
	3	waist behind apron	4	15	11		1 Myelog.
	4	waist in front apron	8	37	29		
	5	inside r. glove	5	5	0		
	6	ant. neck	10	12	2		
	7	r. forearm	8	10	2		
	8	back	12	15	3		
Dr. J. Smith	1	forehead	31	33	2	23 min.	11 G. I.
1-22-54	2	r. shoulder	5	10	5		(4 Spot Films)
	3	waist behind apron	15	19	4		
	4	waist in front apron	37	45	8		
	5	inside r. glove	5	6	1		
	6	ant. neck	12	13	1		
	7	r. forearm	10	12	2		
	8	back	15	17	2		

SUMMARY

1. Permissible dose problems have been discussed.

2. A new simple method for recording radiation dosage received by patients during roentgenologic diagnostic procedures has been described.

3. Methods for recording radiation dosages received by radiologic staff members have been briefly described.

CONCLUSION

We feel that the methods selected for recording roentgen dosage, both for the patient undergoing diagnostic roentgen procedures and by the physician fluoroscopist, are not necessarily ideal. We recognize that other more advantageous methods may be forthcoming. What we have tried to do in this paper is to emphasize and bring into sharper focus the necessity for a permanent recording of roentgen dosage. The primary consideration is the recognition that some such method must be adopted; the actual mechanism is secondary.

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ESTIMATION OF WHOLE-BODY RADIATION DOSES IN ACCIDENTAL FISSION BURSTS*

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TWO nuclear accidents occurred at the Los Alamos Scientific Laboratory, on August 21, 1945, and on May 21, 1946. The accidents centered about the occurrence of uncontrolled fission reactions in such a manner that 10 persons were exposed to bursts of penetrating ionizing radiation. Two of these persons died as a result of exposure to the radiation, while some survivors (Cases 2, 9, and 10) showed clinically insignificant reactions. The case histories of 9 of the individuals have been described in detail by Hempelmann, Lisco, and Hoffman.[4] The following account deals with the methods and physical problems of estimating the radiation doses sustained in these 9 cases. The cases are numbered exactly as they were given in the clinical report.[4]

Penetrating ionizing radiations were the sole cause of injury sustained by the personnel. There were no heat or mechanical effects accompanying the release of energy from the critical assemblies of fissile material. A state of criticality was reached inadvertently and then removed as fast as the reaction time of the operators permitted. There were no complications due to the spread of radioactive fission products, these having been retained within the confines of the assembly. Therefore, the radiation doses were due to the fission process itself and only secondarily due to the delayed radiation from the products of fission.

Injury to tissue was brought about by fast neutrons and gamma rays. The former impart their energy to the tissue primarily by collisions with hydrogen atoms to produce recoil protons. After becoming thermalized, the neutrons are captured. The capture in H^1 is followed by the emission of 2.2 mev. gamma quantum. This gamma radiation produces a "selfdosage" of the tissue, or an "autodose." Neutron capture in N^{14} releases a proton, the reaction having a Q value of 0.6 mev. Of particular interest are the induced radioactivities in Na^{24} and P^{32} which follow neutron capture in Na^{23} and P^{31}. These activities occurred in measurable quantities, as is shown in Table 1 (see also Reference 4). The Na^{24} activity provided a physical means by which neutron doses could be estimated.

The gamma rays contributing to the tissue dose were: first, the prompt gamma rays released in the fission process; second, the autodose arising from H^1 captures, as mentioned above; and, third, the delayed gamma rays coming from the fission products remaining after the fission burst. Dosage due to delayed gamma radiation depends, of course, on whether or not the individual remains near the fissionable assembly after the fission burst has occurred. Only in Case 1 below did the delayed gamma radiation assume importance, since that individual remained close to the setup after the critical state had come and gone.

The major components of the complex radiation dose are listed in order of magnitude: (a) fast-neutron collisions producing recoil protons in tissue, (b) hydrogen-capture gamma rays producing an autodose of the whole body, (c) slow-neutron captures in N^{14}, and (d) prompt gamma rays from the fissioning material. Induced activities in Na, Cl, P, and Mn give rise to a negligible tissue dose. However, an important cause of tissue injury, which appeared in two operators close to the fissioning assembly, was the high intensity of soft radiation in the blue glow. This comprised secondary soft x-rays, slow neutrons, and

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TABLE I

PHYSICAL DATA ON PERSONNEL

Case	1 Distance from Assembly, cm.	2 Height, cm.	3 Body Weight, kg.	4 Estimated Torso Thickness,* cm.	5 P^{32} in Urine,† dps	6 Na^{24} in Urine, dps
1	30	179	92.0	30	110.00	22.50
2	300	170	86.3	30	4.70	1.38
3	50	168	59.4	20	357.00	248.00
4	100	175	79.4	25	68.10	15.10
6	140	165	68.0	25	13.60	7.75
7	210	183	78.5	22	2.97	5.35
8	210	178	74.8	22	2.53	3.35
9	400	178	77.1	25	1.40	1.92
10	400	168	59.09	20	1.40	1.27

* Anteroposterior thickness at diaphragm.
† Specific activity in disintegrations/sec./mg. normal element.

secondary electrons, most of which are absorbed in superficial tissue layers.

I. INDUCED RADIOACTIVITY

The detailed protocol on the induced radioactivities in the bodies of the patients has been published (Reference 4, Section VIII), and the results have been discussed in extenso. Here are given the specific activities of P^{32} and Na^{24} in urines (Table I, columns 5 and 6) to show one of the effects of thermalized neutrons in the body. It turned out that the urine activities were not as selfconsistent as were the serum Na^{24} activities. For purposes of dose estimation the serum Na^{24} activities given in Table II, column 1, appeared to give a representative measure of neutrons incident on the body tissues.

It is assumed that the Na^{24}-serum activity measures the average slow-neutron flux throughout the body. Using an average value of serum Na^{23} of 3.25 mg./cc. and a capture cross section of 0.45 barns for Na^{23}, the slow-neutron flux is given by 6.48×10^{9} times Na^{24} specific activity and is tabulated in Table II, column 2. For the computation of hydrogen captures of neutrons, the capture cross section is taken as 0.31 barns and the density of H^{1} atoms/gm. of tissue as 6.6×10^{22}. The captures in N^{14} are based on a capture cross section of 1.75 barns, and that N^{14} represents 3 per cent by weight of tissue.

The captures in tissue elements shown in Table II are not produced uniformly through the bodies of the personnel. The primary fast neutrons which gave rise to the slow neutrons are assumed to have had an average energy of 0.5 mev. Neutrons of

TABLE II

SLOW-NEUTRON FLUX AND CAPTURES BASED ON SERUM Na^{24} SPECIFIC ACTIVITY

Case	1 Na^{24} in Serum* dps	2 Slow-Neutron Flux, n† $\times10^{9}$/cm.	3 Hydrogen Captures/gm. $\times10^{9}$	4 N Captures/gm.‡ $\times10^{8}$
1	18.00	117.0	2.400	2.660
2	1.10	7.1	0.145	0.161
3	73.60	477.0	9.750	10.820
4	13.30	86.3	1.760	1.960
6	7.10	46.1	0.942	1.040
7	3.80	24.7	0.510	0.561
8	2.03	13.2	0.270	0.300
9	1.54	10.0	0.200	0.230
10	1.22	7.9	0.160	0.180

* Specific activity of Na^{24} in disintegrations/sec./mg. serum sodium.
† Slow-neutron flux = $6.48\times10^{9}\times$ specific activity Na^{24}.
‡ N^{14} captures = $2.27\times10^{-3}\times$n. Hydrogen captures/gm. = 0.0204.

this energy administered over a large area of skin have low penetrating power. The curves of Tait[19] and of Marshak[7] show a reduction to half intensity at about 2.8 cm. depth in aqueous tissue. This is also seen in the calculations of Biram.[1] There is, however, no specific evidence to support the assumption of 0.5 mev. as the average neutron energy. The assumption is based solely on the knowledge that there was sufficient scattering material around the assemblies to ensure at least one collision for most of the fast neutrons.

The neutrons represented in Table II, as measured by serum Na^{24} activity, probably diffused throughout 50 per cent of all the tissues of the body. The diffusion length for thermal neutrons is about 2.9 cm. Diffusion coupled with the maximum penetration of fast neutrons to 8 cm. accounts for half the body tissues. Workers with water baths for thermalizing fast neutrons usually allow 3 to 4 cm. for the diffusion loss of thermal neutrons at the edges of the bath.

By the same token, however, there is the problem of estimating the loss of thermal neutrons from the body. The edge effect arising in 3 cm. is appreciable, since 3 cm. is a significant distance compared to the cross-sectional dimensions of all parts of the body, including the torso. The diffusion loss, along with the loss of scattered fast neutrons and their effect on the estimated tissue dose, can be assessed only by means of judicious guesses until experimental data become available. The captures shown in Table II form the basis for the minimum possible doses. Corrections for diffusion and scattering loss of neutrons are discussed later in this report (Section VII).

II. THE AUTODOSE DUE TO HYDROGEN-CAPTURE GAMMA RADIATION

At the time of the accidents in 1946 the methods for assessing autodosage had not been fully developed in the literature. In order to calculate a minimum possible value for this kind of dose, the reciprocal relationship between a radiation source and absorber was used. The reciprocity theorem is discussed by Mayneord[9] and by Bush.[2] Calculations were made using the approximation formulas for the integral dose in ellipsoidal cylinders as developed by Mayneord[9] with the appropriate corrections for elongation, namely, the ratio of the length to the width. Integral doses were computed for the uniform distribution of hydrogen captures at a density of 10^8/gm. of tissue. The linear absorption coefficient of the gamma rays was 0.028 $cm.^{-1}$, and the source strength was taken as 7.43×10^{-3} r/10^8 H^1 captures.

It turned out, as will be seen in the following, that estimates based on these calculations were low, because the contribution of one part of the body to the other was not accounted for. The calculation for each part, as for example the torso, gave only the dosage arising from the source material within that part. Experiments on a mock-up of a standard man, filled with Na^{24} in water, showed that the first computed minimum values of integral dose were 50 per cent of the experimentally determined values. With the subsequent publication by Bush[2] of the integral autodose calculations for over-all body dosage, taking into account the exposure of all parts by the others, it was found that the first estimates were 57 per cent of his theoretical values.

In the following there will be described the experimental method using a mock-up man to determine the integral dose of hydrogen-capture gamma radiation. This will be followed by the theoretical computation based upon Bush's application of the reciprocity theorem to the problem.

A mock-up man was constructed of 0.030 in. copper sheet (0.762 mm.) to weigh 70.072 kg. when filled with water. The masses of the various parts of the man are shown in Table III. The man described by Mayneord,[10] weighing about 72 kg., served as a prototype for this man. (Compare also the data of the International Commission on Radiological Protection[8] and Lisco's figures, quoted by Schubert,[17] for the properties of the Standard Man.) The purpose was to dissolve $Na^{24}Cl$ in the water of the

Table III

GAMMA-DOSE RATES AT ANTERIOR SURFACE OF 70-KG. WATER MOCK-UP MAN WITH 80,978 DISINTEGRATIONS Na^{24}/MIN./CC.

Part of Man	M, Mass of Parts, kg.	I,* Gamma Intensity, mr/hr.	MI, Integral Dose Rate, gm.r/hr.
Foot (lower)	1.388	29.4	40.7
Knee	8.180	38.7	317.0
Thigh	10.200	59.2	603.0
Diaphragm	36.100	68.3	2470.0
Arms (elbow)	5.620	60.3	339.0
Neck	4.330	46.5	201.3
Head	4.250	46.5	197.5
Total	70.072		4168.5

Height of mock-up man = 168 cm.
Intensity at center of torso (at diaphragm) = 123 mr/hr.
4168.5 gm. r/hr. = 69.5 gm.r/min.

* Gamma intensity multiplied by 0.526 to correct for energy dependence. The average dose rate is 95.7 mr/hr. or 77 per cent of central dose rate.

filled mock-up man and measure the gamma ray dose rates in and about the parts of the body. The two gamma rays from Na^{24}, having energies of 1.38 mev. and 2.76 mev., were assumed to give a good approximation to the 2.2 mev. H^1-capture gamma rays. The gamma ray dose rates for a known number of Na^{24} disintegrations/cc. of mock-up water would serve as a basis for estimating the autodose due to H^1 neutron captures.

Samples of Na^{24} Cl in the Los Alamos "water-boiler" reactor for an hour at the 5 kw. level were measured for their gamma and beta activity. The following table gives the results obtained with three different gamma-detecting meters along with the beta activity:

All meters were calibrated against radium in 0.5 mm. Pt capsule.

The output in gamma r/mc.hr./cm.^{2} at 1 cm. is the constant k of Mayneord[11] and the I_γ of Marinelli, *et al.*[6] This fundamental constant has the value 19.3 r/mc.hr. for Na^{24}. More recently Mayneord[12] quotes measurements as yielding a value of 18.85 r/mc.hr. At the time the above measurements were made, it was assumed that the constant was near 20 r/mc.hr. The table above shows that the counter-type Victoreen survey meter, which had a thin-walled Geiger-Müller tube mounted inside a steel casing, gave over twice the expected gamma intensity. This was attributed to variation in response with gamma energy. This counter meter agreed exactly with the brass-walled, direct current, ionization-chamber-type meter in readings made about the mock-up man.

It was assumed that the air-wall-type chamber gave the correct gamma intensity in roentgens, namely, 23.1 r/mc.hr. This chamber was not suited for practical measurements about the mock-up man. Therefore, the non-air-walled instruments were used and their readings reduced in the ratio of $23.1/44.1 = 0.526$, where 44.1 is the average of 41.6 and 46.6.

The autodose rates found in and about the mock-up man are shown in Table III. At the anterior diaphragm surface the gamma intensity on the counter-type survey meter was 130 mr/hr. for 80,978 Na^{24} disintegrations/min./cc. This was corrected to the air-wall chamber equivalent reading by multiplying by 0.526, as were all the intensities in Table III. The integral dose rate measured externally at the diaphragm is seen to be 69.5 gm.r/min., which becomes

DETERMINATION OF GAMMA-ROENTGEN EMISSION OF Na^{24}

Meter	NaCl Weight, gm.	Total Beta Radiation, mc.	Gamma Ray Intensity, mr/mc.hr./cm.^{2} at 1 cm. distance
Victoreen Geiger-Müller counter survey meter	10	2.15	41.6
Brass-walled ionization chamber ("Wattsmeter")	10	2.14	41.6
Victoreen "air-wall" 0.25 r chamber	10	2.14	23.1
Victoreen Geiger-Müller counter survey meter	1	0.264	46.6

$69.5/80{,}978 = 8.6 \times 10^{-4}$ gm.r/Na^{24} disintegration. Since the dose rate on the central axis of the torso is 123 and the external dose rate is 68.3 mr/hr., the average is 95.7 mr/hr. This is perhaps high in that the difference between the central and external surface-dose rates is not as great in the lower limbs as was found in the torso.

The average body-dose rate of 95.7 mr/hr. for 80,978 Na^{24} disintegrations/min./cc. forms the basis for computing the average gamma ray (2.2 mev.) dose throughout the body. The average body dose becomes 1.97×10^{-5} mr/Na^{24} disintegrations/cc., which is converted to H^1-capture quanta by multiplication by the ratio of the k values, namely, 9.9/19.3, where 9.9 r/mc.hr./cm.^{2} at 1 cm. is the k value for H^1-capture gamma rays. This leads to the average dose rate of 1.05×10^{-5} mr/H^1-capture/cc. Table IV, column 1, shows the gamma ray dose in r obtained by the product of hydrogen captures/gm. and the average dose rate, corrected in the ratio of the body weight to 70 kg.

TABLE IV

INTEGRAL AUTODOSES DUE TO HYDROGEN-CAPTURE GAMMA RAYS

Case	Mock-up Man Measurements		Theoretical Estimate	
	Average Body Dose, r	Integral Auto-Dose,* megagm.r	Autodose Rate for Radium,† megagm.-r/mc.hr.	Theoretical Integral Autodose,† megagm.r
1	33.0	1.91	104	2.220
2	1.9	0.108	99	0.128
3	87.0	5.01	59	5.150
4	21.0	1.20	88	1.380
6	9.5	0.55	73	0.613
7	5.9	0.35	85	0.380
8	3.0	0.18	80	0.190
9	2.3	0.13	84	0.150
10	1.4	0.08	59	0.085

* Computed from the mock-up man constant 6.06×10^{-4} gm.r/H^1-capture.

† Computed from the theoretical tables for radium autodosage of Bush.[2] Column 3 gives integral dose for 1 mc.hr. of Ra/gm. of tissue.

The average integral dose rate for the entire body becomes $(95.7/68.3) \times (8.6 \times 10^{-4}) = 11.8 \times 10^{-4}$ gm.r/Na^{24} disintegrarions/cc. and the integral autodose rate is $11.8 \times 10^{-4} \times 0.513 = 6.06 \times 10^{-4}$ gm.r/H^1-capture.

Integral autodoses based upon this constant are shown in Table IV, column 2. To illustrate the computation, take Case 3 in which the hydrogen captures (Table II) were 9.75×10^9/cc. The body weight is divided by 70 kg. to normalize it against the mock-up man: $59.4/70 = 0.849$. The integral dose is then $0.849 \times 9.75 \times 10^9 \times 6.06 \times 10^{-4} = 5.01 \times 10^6$ gm.r, or 5 megagm.r. The comparison by weight with the mock-up man figures is approximate; it does not take into consideration variations of height and thickness of the individuals.

For a comparison with the theoretical values based upon Bush's[2] computation of integral autodose, Table IV, column 4, shows the integral autodoses, assuming that the doses as computed by Bush for radium can be converted to H^1-capture gamma ray dosage according to the ratio of k values (see, for example, Mayneord,[11] p. 154). The k value for radium in a 0.5 mm. Pt container is 8.3 r/mc.hr. For H^1-capture gamma rays k is 9.9 r/mc.hr., or 9.9 r/1.332×10^{11} r/capture. From Bush's paper the values for integral autodose rates for radium in patients of varying heights and weights are given in Table IV, column 3. As an example of the arithmetic, take Case 3. The radium autodose rate, column 4, is 59 megagm.r/mc.hr., this having been determined from Bush's table by Case 3's weight of 59.4 kg. and 168 cm. height, as given in Table I. The radium autodose rate is corrected for radiation quality by the ratio 9.9/8.3, and then multiplied by the H^1-captures/cc. and divided by 1.331×10^{11}. Thus, $59 \times (9.9/8.3) \times 9.75 \times 10^9 / 1.331 \times 10^{11} = 5.15$ megagm.r.

The autodoses computed thus far are not minimum doses due to capture gamma rays; they are high because the H^1-captures did not occur uniformly throughout the body, but rather were concentrated toward the

side facing the neutron source. In Section VIII below the distance factor for the source of radiation is discussed.

III. HEAVY-PARTICLE DOSE

Fast-neutron Recoils. The estimation of fast-neutron dose is based on the assumption that each neutron captured in H^1 released 0.5 mev. to the tissue before thermalization and that 1 roentgen-equivalent-physical (rep) corresponds to 93.1 ergs /gm., or 5.8×10^{13} electron volts/gm. Thus, each captured neutron/gm. contributed $0.5\times10^{6}/5.8\times10^{13}=8.62\times10^{-8}$ rep. This constant, multiplied by the number of neutron captures in H^1/gm. of tissue, gives the dose in rep which is tabulated in column 1, Table V. The number of H^1 captures is taken from column 3, Table II. Although the dose is designated as fast-neutron dose, it comprises energy released in the form of recoil protons and heavier atoms.

N^{14}-Neutron Captures. The dose arising from N^{14} capture of slow neutrons is a heavy-particle dose. The reaction Q value is 0.6 mev. A proton and recoil C^{14} are the heavy particles which cause ionization in tissues; for this reason the N^{14}-capture dose is tabulated with the fast-neutron dose in Table V. The dose in rep is calculated on the assumption that each capture releases 0.6 mev. to the tissue, and that 1 rep equals 5.8×10^{7} mev./gm. of tissue. The rep shown in column 2, Table V, are based on the N^{14} captures given in Table II, column 4. (For a discussion of the nature of the N^{14}-capture dose, the reader is referred to Solomon,[16] Chapter 4.)

Recoil Deuterons. In the 1948 report[5] it was pointed out that the deuteron recoil following H^1-neutron capture has a kinetic energy of 1,300 ev. For 10^9 H^1 captures /gm. of tissue this recoil contributes 0.022 rep. From Table II, column 3, it is seen that the H^2-recoil dose is negligible compared to the H^1-capture gamma ray autodose, Table IV, column 1, even if the relative biological effect (RBE) factor is taken as 10 for the H^2 rep.

TABLE V

HEAVY-PARTICLE DOSES: FAST-NEUTRON RECOILS AND NEUTRON CAPTURES IN N^{14}

Case	1 Fast-Neutron Dose rep	2 N^{14} Dose Neutron Capture rep	3 Total of Fast-Neutron and N^{14} Doses rep	4 Total Body Dose* 10^6 rep
1	20.7	2.74	23.44	2.16
2	1.3	0.17	1.47	0.13
3	83.6	11.15	94.75	5.63
4	15.1	2.02	17.12	1.36
6	8.1	1.07	9.17	0.62
7	4.4	0.57	4.97	0.39
8	2.3	0.31	2.61	0.20
9	1.7	0.24	1.94	0.15
10	1.4	0.18	1.58	0.093

* Total body dose is the product of column 3 multiplied by body weight.

IV. DOSE OF PROMPT AND DELAYED GAMMA RADIATION

In order to estimate the dose of the prompt gamma rays from the fissions, measurements were made of the variation of delayed gamma ray intensity from the assembly, to which Cases 3 to 10 were exposed. Over the range of distances (r) from 75 cm. to 2,500 cm. the gamma ray intensity fell off as $1/r^{1.6}$ rather than as $1/r^2$. The deviation from the $1/r^2$ law was also found in the planted film badges which showed a falling-off as $1/r^{1.54}$ for an exposure which included the prompt as well as some of the delayed gamma rays. By way of comparison, the urine-induced activities in P^{32} and and Na^{24} fell off as $1/r^{2.4}$, while the serum Na^{24} went as $1/r^{2.3}$. The distances of personnel are given in Table I and their Na^{24} activities in Tables I and II.

In the above mentioned assembly for Cases 3 to 10, it was estimated that at a 1 meter distance the maximum amount of prompt gamma radiation to emerge would lead to a dose of 7.2 r. On the basis of the observed variation with distance r as $1/r^{1.6}$, the incident gamma ray dose on personnel can be estimated using the distances shown in Table I. In Table VI, column 1,

are given the prompt gamma ray doses at the skin surface.

Assuming an average energy of 1 mev. for the gamma rays, for which the linear absorption coefficient in water is 0.03 $cm.^{-1}$, the average body dose is calculated from Mayneord's[10] tables and tabulated in column 2. For example, Case 4, at 100 cm. from the source of radiation and having a torso thickness of 25 cm., had an average body dose of 0.732 r/incident r due to plane-wave radiation. (The factors are tabulated in Table IX.) This is corrected for a source at 100 cm. by the factor 0.85. The resultant average body dose is $7.2 \times 0.732 \times 0.85 = 4.5$ r. The prompt gamma ray doses are seen to be small.

Cases 3 to 10 had no significant body dose due to delayed gamma radiation. This group of individuals left the vicinity of the assembly at about 20 seconds after the fission burst. Cases 1 and 2, however, did not leave their positions after the burst. Case 2 was far enough away (300 cm.) so that his dose due to prompt and delayed gamma radiation was negligible. The particular assembly near which he was stationed was heavily shielded. On the other hand, Case 1 was near that assembly (30 cm.) and worked with it during and after the fission burst. On the basis of his account of his protracted manipulations of the assembly, it is estimated that he sustained a minimum average body dose of 220 r of gamma radiation.

Gamma ray measurements on a similar assembly showed a dose rate of 190 r/min., measured in an air-walled chamber, at 30 cm. distance at 10 seconds after the burst. In the earlier reports[4,5] Case 1's minimum was stated as 110 r, which is now regarded as far too low in view of the fact that the patient is known to have worked in and about the setup for at least 10 minutes after the fission burst occurred. The 220 r includes prompt and delayed gamma radiation dose (Table VI).

V. WHOLE-BODY DOSE DUE TO INDUCED RADIOACTIVITIES

Although the activities induced by slow-neutron capture in tissue elements such as Na^{24} were measurable, their contribution to radiation dose was very small. It turns

TABLE VI

PROMPT AND DELAYED GAMMA RAY DOSES AND SOME REPRESENTATIVE AMOUNTS OF INDUCED ACTIVITIES IN PERSONNEL

Case	1	2	3	4	5	6	7	8
	Prompt-Gamma Ray Dose at Skin, r	Prompt-Gamma Ray Dose, Body Average, r	Body Average, Delayed-Gamma Ray Dose, r	Total Amount of Induced Activity in Microcuries				
				Na^{24}	K^{42}	Mn^{56}	Cl^{38}	Mg^{27}
1								
2			220	65.0	15.7	8.6	72	2.15
3	18.1	11.4		170.0	41.0	22.7	191	5.70
4	7.2	4.5		41.0	10.0	5.4	46	1.36
6	3.8	2.4		18.8	4.5	2.5	21	0.60
7	1.8	1.3						
8	1.8	1.3						
9	0.7	0.5						
10	0.7	0.5						

For Case 2 the prompt and delayed-gamma ray doses were negligibly small.
For Case 1 the prompt and delayed doses are lumped together in the delayed gamma ray dose.
Amounts of induced activity can be corrected for possible slow-neutron loss by the factor 1.18.

out that the dose is small compared to the autodose arising from H^1-capture gamma rays.

For purposes of indicating the magnitude of induced activity and its resultant dose, the following computations for Case 3 are typical. Referring to Table II, column 1, the specific activity, Case 3, is 73.6 dps./mg. serum Na^{23}. For an Na-content of 3.25 mg/cc. of serum, this activity is 6.47×10^{-3} μc Na^{24}/cc. The beta rep due to the total disintegration of this amount of Na^{24} is calculated on the basis of Marinelli's[6] evaluation of the constant: 29 beta rep/μc Na^{24} destroyed. The 6.47×10^{-3} μc/cc. of serum then deliver 0.187 rep over a period of several half-lives of Na^{24} (half-life, 14.8 hours). With a hematocrit of 40 per cent this dose becomes $0.187 \times 0.6 = 0.112$ rep/cc. of blood.

The average beta and gamma ray dose throughout the body is best taken on the basis of the total Na in the body and an average amount/cc. of tissue computed. Total amounts of Na are quoted as: 63 gm./70 kg. of man, Shohl;[18] 89.2 gm./66 kg. of man, Moore;[13] and 105 gm. in the Standard Man weighing 70 kg. (International Commission on Radiological Protection[8]). The more recent figure of 105 gm. becomes 1.5 mg. Na/gm. of tissue averaged throughout the body. On the basis of Case 3's specific activity, it turns out that for his 59.4-kg. weight he had 170 μc Na^{24} total. For comparison, the permissible total in a 70 kg. man is 15 μc.[8] The average beta ray dose in Case 3 is 0.083 rep.

The gamma ray dose delivered by the Na^{24} is an autodose like that due to H^1-capture gamma rays. To compute the gamma ray dose rate in Case 3, take an average tissue Na content of 1.5 mg./gm., specific activity of 73.6 Na^{24} dps./mg. Na^{23}, and the average body-dose rate for a 70-kg. man found in Table III, namely, 1.97×10^{-8} r/Na^{24} disintegrations/gm. of man. There are, then, 3×10^{-3} μc Na^{24}/gm. for which the total number of disintegrations is 8.45×10^6, for a mean life of Na^{24} of 21.3 hours. The average body gamma ray dose during the destruction of Na^{24} is 0.14 r. The biologic half-life of Na^{24} of 12 days plays no significant part in this dosage.

The beta and gamma ray doses toegther comprise less than 1 per cent of the H^1-capture gamma ray dose recorded in Table IV. For Case 3 the H^1-capture gamma ray dose is 87 r. The Na^{24} beta dose is 0.083 rep and the gamma ray dose 0.14 r. Since the Na^{24} doses are small, they are not tabulated.

The possible contributions to the induced activity dose due to short-lived isotopes other than Na^{24} were examined. The isotopes K^{42} (12.4 hours), Mn^{56} (2.6 hours), Cl^{38} (0.62 hours), and Mg^{27} (0.16 hours) were the chief ones concerned. The total amounts of isotope along with Na^{24} are tabulated in Table VI for Cases 1, 3, 4, and 6. Taking account of the isotopic abundance, the amount in the body, and the energies of the radiations, the doses from the four isotopes other than Na^{24} are negligibly small. The magnitudes of their contributions decrease in the following order: Cl^{38}, K^{42}, Mn^{56}, and Mg^{27}. The gamma ray doses are in the same order totaling an additional 140 per cent of the Na^{24} average whole-body dose.

VI. ESTIMATE OF GAMMA RAY DOSE

The total induced activities shown in Table VI along with the serum Na^{24} specific activities in Table II provide an indication of the order of gamma ray intensity from the bodies of victims to be expected at various dose levels. The gamma ray intensity has an advantage in that it can be measured at the anterior diaphragm level. The assay of activity in sera or in urines is more elaborate in that it requires a careful preparation of samples and counting. The body elimination of P^{32} has yet to be clarified.[4] Sodium in urine is subject to wide fluctuations. Therefore, the sera Na^{24} are the most reliable measure of neutron flux.

Case 3 in Table VI had a total of 170 μc Na^{24} which was mostly produced in the arms and torso, since he was close to the source of neutrons. His dose was lethal as

was that of Case 1, although the latter had only 65 μc Na^{24}. The additional dose due to delayed gamma radiation in Case 1 probably was a major factor in his death. For purposes of estimating the amount of Na^{24} which would indicate a lethal dose, it is assumed that the activity of Na^{24} of 18 dps. in Case 1 was less than that corresponding to a lethal dose such as in Case 3 with a serum specific activity of 73.6 dps. This is substantiated to some extent by the fact that Case 4 had a specific activity of 13.3 dps. and survived.

The mock-up man's measurements showed 130 mr/hr. for a concentration of 80,978 Na^{24} disintegrations/min./cc. measured at the diaphram skin surface with the counter-type survey meter (calibrated against Ra), not corrected for the Na^{24} gamma energy. In terms of counts, the same gamma-meter showed 4,500 cpm. /1 mr/hr., or 72 counts/disintegration/cc. of mock-up man. The latter figure shows that 1 gamma ray in every 2,000 emitted in the man was recorded by the counter. If measurements had been made on Case 1 at the time of the accident, an approximate counting rate of about 12,500 cpm., or 2.8 mr/hr., at diaphragm level, would have been recorded due to the Na^{24} alone. The biological half-life in man for Na^{24} of about 12 days makes it necessary to consider only the natural decay for the decrease in intensity with time.

The essential feature of these data is that the Na^{24} gamma rays occur in intensity sufficient for an appraisal to be made of the magnitude of the radiation exposure. There are three aspects of this gamma assessment of dose which need to be pointed out. The first is the correction for the natural decay of Na^{24} according to a 14.8 hour half-life. The second is that the other short half-lived isotopes such as K^{42}, Mn^{56}, Cl^{38}, and Mg^{27} be allowed for. The Mn^{56}, Cl^{38}, and Mg^{27} will have decayed after about 12 hours. After that the K^{42} will continue to contribute about 25 per cent of the gamma reading, since it has a half-life of 12.4 hours. The third and most important qualification on the gamma assay of dose is that it gives a measure only of thermal neutrons in the body. The question as to the spectrum of fast neutrons which gave rise to the thermal neutrons has to be estimated from other information about the exposure.

VII. CORRECTIONS TO THE MINIMUM DOSES

The various components contributing to the total dose are summarized in Table VII under the column entitled "Primary Doses." For illustrating the make-up of the total dose, Case 6 was found to be most suitable. Other cases near the source involved extraneous factors obscuring the fission-burst dose. Case 1 was near a heavily shielded source and, moreover, sustained a large dose of delayed gamma radiation. Case 3 was too near the source to permit accurate evaluation of the distance relative to the source, since his hands were on the assembly and his feet about 90 cm. from its

TABLE VII

SUMMARY OF AVERAGE BODY DOSE COMPONENTS FROM FISSION BURST

Case 6. Average Neutron Energy 0.5 mev.

Component	Primary Doses	Equivalent-Gamma Doses,* r	Equivalent Doses as Percentage of Total
H^1-capture gamma ray (Table IV)	9.5 r	9.5	16.4
Fast neutron (Table V)	8.1 rep	40.5	70.1
N^{14}-neutron capture (Table V)	1.07 rep	5.4	9.3
Prompt gamma rays (Table VI)	2.4 r	2.4	4.2
Total		57.8	100.0

These doses are not corrected for slow-neutron loss or fast-neutron scatter.

* One H^1 rep is biologically equivalent to 5 gamma rep.

center. Case 4 is known to have been partly shielded from the assembly by Case 3's body. Case 6 was entirely unprotected; his entire body, except possibly his lower legs, was exposed to the source at a reasonably well-ascertained distance of 140 cm.

For a comparison of the relative magnitudes of the components, a relative biological effectiveness (RBE) of 5 is assigned to the H^1 rep. The RBE has been described in the clinical case report[4] (Sections II *C* and IV *A*). Zirkle (Oberlin Conference,[16] p. 53) takes as a rough average RBE = 4 for protons. On the other hand, Neary[11] takes RBE = 10 in tolerance-level considerations. In Table VII the column called "Equivalent Gamma Dose" has the proton rep multiplied by 5. The last column shows their relative magnitudes. For 0.5 mev. neutrons the recoil rep have already become 70 per cent of the total dose.

The dose components in Table VII are minimum doses. The dose components due to H^2 recoils, slow-neutron-induced activities, and delayed gamma rays have been omitted because they are negligibly small. The dose due to neutron-capture gamma rays from surrounding objects, walls, floors, etc., has not been considered. An important item in considering these as minimum doses is loss of thermal neutrons by diffusion from the body as was mentioned earlier in this report. The first three components in Table VII are directly proportional to the Na^{24}-induced activity in the blood sera.

In considering a possible correction for diffusion loss of slow neutrons, the dimensions of the various parts of the human body are important. The largest part, the torso, is about 20 to 25 cm. thick and 30 to 40 cm. wide, from which diffusion loss is conservatively estimated to be 10 to 15 per cent.

The other body members, such as head (15 × 20 × 18 cm.), arms (10 cm. diameter) and thighs (15 cm. diameter), present blocks of aqueous material from which diffusion losses range from 20 to 30 per cent, depending on thickness. As a final estimate of slow-neutron loss due to diffusion, 15 per cent appears reasonable and conservative. Thus, the first three dose components in Table VII should be increased by a factor of 1.18 to account for slow-neutron loss.

The energy of the primary neutrons not only influences the amount of leakage of slow neutrons from the body, but obviously plays an important part in the dosage. In Cases 1 and 2 the assignment of an average energy of 0.5 mev. to the neutrons is justifiable because of the heavy shielding about the critical assembly to which they were exposed. In the remaining 7 cases there is the possibility that a small fraction of the neutrons may have had high energies. As Glasstone and Edlund[3] point out, the fission spectrum of neutrons extends from about 0.1 mev. out to beyond 10 mev., the majority of neutrons having energies in the range 1 to 2 mev.

The pronounced contribution of fast neutrons to the dose is illustrated by the following example which serves also to estimate a maximum possible dose to be added to the computations made thus far. If 5 per cent of the neutrons indicated by Na^{24} activities have an average energy of 5 mev. rather than 0.5 mev., then the total fast-neutron rep in Table VII and Table V, column 1 are increased by 45 per cent. Thus, in Cases 3 to 10 a reasonable maximum dose due to neutron energy in the fast-neutron rep would require that the minimum doses be increased by a factor of 1.45.

There remains yet a correction to be made for fast neutrons scattered from the body before becoming thermalized. This correction applies only to the fast-neutron component, since it implies that a fast neutron imparted a fraction of its energy to the tissue in the form of a recoil atom and then was scattered out of the body. This is a dosage in the form of fast-neutron rep which is tentatively assumed to be 10 per cent of the total.

To summarize the corrections which have been described in the foregoing paragraphs: The first is an 18 per cent increase in the

TABLE VIII

SUMMARY OF AVERAGE BODY DOSES

Case	1	2	3	4	5	6
	Capture Gamma Ray, r	Fast Neutron, rep	N^{14} Capture, rep	Prompt Gamma Ray, r	Gamma-Equivalents	
					Average Dose, r†	Incident Dose, r‡
1	33.0	27.0	3.23	220.0*	404	587
2	1.9	1.7	0.20		11	16
3	87.0	109.0	13.20	11.4	709	1125
4	21.0	19.6	2.38	4.5	136	219
6	9.5	10.5	1.26	2.4	71	110
7	5.9	5.7	0.67	1.3	39	56
8	3.0	3.0	0.37	1.3	21	30
9	2.3	2.2	0.28	0.5	15	22
10	1.4	1.8	0.21	0.5	12	17

Doses in this table are corrected for slow-neutron diffusion loss and fast-neutron scatter.
* Prompt and delayed doses are combined in this one case.
† In the summation of doses in columns 1, 2, 3, and 4 the heavy-particle doses were multiplied by the RBE = 5.
‡ 1 mev. gamma rays for which $\mu = 0.03$ cm.$^{-1}$.

first three components of dose listed in Table VII, since these are proportional to the slow-neutron flux. The second correction is a 10 per cent increase in the fast-neutron component to compensate for fast-neutron scatter out of the body. The average body doses, taking these corrections into account, are summarized in Table VIII, columns 1, 2, 3, and 4. In the same Table, column 5, are the average body doses which are the gamma equivalent of the sum of components listed in the first four columns. It should be emphasized that the average body doses of Table VIII are averages of dose within the tissue and are not to be confused with average incident dose to the body.

By means of the conversion factors in

TABLE IX

CONVERSION FACTORS FOR EXPRESSING AVERAGE BODY DOSES AS INCIDENT 1 MEV. GAMMA AND 80 KEV. ROENTGEN-RAY DOSES*

Case	Source-to-Body Distance Correction		Average Body Dose/r†		Corrected Average Body Dose/r	
	Gamma	Roentgen Ray	Gamma	Roentgen Ray	Gamma	Roentgen Ray
	1	2	3	4	5	6
1	0.75	0.78	0.69	0.23	0.52	0.28
2	0.94	0.96	0.69	0.23	0.65	0.22
3	0.80	0.83	0.78	0.36	0.63	0.30
4	0.85	0.91	0.73	0.30	0.62	0.27
6	0.88	0.94	0.73	0.30	0.64	0.28
7	0.92	0.96	0.76	0.33	0.70	0.32
8	0.92	0.96	0.76	0.33	0.70	0.32
9	0.94	0.97	0.73	0.30	0.69	0.29
10	0.94	0.97	0.78	0.36	0.73	0.35

All entries in this table are in units of r/incident r.
* Assuming the radiating assembly is a point source.
† For incident plane wave.

TABLE X

INTEGRAL DOSES IN TERMS OF ENERGY ABSORBED CONVERTED TO EQUIVALENT GAMMA ROENTGENS

	1	2	3	4	5
Case	Heavy-Particle Dose,* megagm.-r	Capture-Gamma Dose,† megagm.-r	Prompt-Gamma Dose,‡ megagm.-r	Delayed-Gamma Dose, megagm.-r	Total Integral Dose, megagm.-r
1	13.8	2.25		20	36.0
2	0.8	0.13			0.93
3	36.2	5.90	0.68		42.8
4	8.8	1.30	0.36		10.7
6	4.0	0.65	0.16		4.8
7	2.5	0.41	0.11		3.0
8	1.3	0.21	0.10		1.6
9	1.0	0.15	0.04		1.2
10	0.6	0.09	0.03		0.73

* From Table v, columns 1 and 2, corrected for RBE = 5, and neutron losses.
† From Table iv, corrected for slow-neutron loss (18 per cent).
‡ From Table vi, column 2, multiplied by patient's weight.

Table ix, the average body dose is transformed into incident gamma dose shown in column 6, Table viii. These are the incident doses coming from the assembly (assumed to be a point source) which would produce the body average dose in column 5.

The total integral dose as a sum of the component integral doses is given in Table x, column 5. This is a gamma integral dose in that the heavy-particle dose was converted to the biological equivalent by the RBE factor of 5. The calculation is made by multiplying the average body dose by the body mass to give the integral dose.

VIII. SOFT ROENTGEN RAY EQUIVALENT OF THE HEAVY-PARTICLE DOSE

The low penetrating power of 0.5 mev. neutrons was pointed out in Section II above. The induced activities ultimately measured in the sera were concentrated at the instant of exposure in the half of the body facing the source of neutrons. The computations of the major dose components listed in Table viii are on the basis of average values throughout the body, because the Na^{24} became uniformly distributed throughout the body. Actually, the fast-particle doses, namely, the fast-neutron and N^{14}-capture doses, were initially concentrated along the surfaces of the body facing the source.

Therefore, the conversion of the dose to gamma-ray equivalents, as shown in columns 5 and 6 of Table viii, and in Table x, column 5, is somewhat misleading as regards the physical picture of how the dose was administered. A closer approximation to the distribution of the heavy-particle dose due to the 0.5 mev. neutrons is obtained by converting it to 80 kev. roentgen ray dosage for which the linear absorption coefficient is $\mu = 0.14$ cm.$^{-1}$ and the half-value layer in copper is 0.082 mm. A comparison of the depth-dose data for the 80 kev. roentgen rays with the theoretical wide-beam depth dose for 0.5 mev. neutrons as published by Tait[19] suggests that the dose distribution in whole-body irradiation would be similar for the two radiations.

Table ix shows the factors which enter into the conversion of the heavy-particle rep in Table viii, columns 2 and 3, into an equivalent incident dose of 80 kev. roentgen rays. The heavy-particle average body dose for Case 3 totals 122.2 rep, which comprises the H^1-recoil dose corrected by the factor 1.3 for neutron loss and scatter, and the N^{14}-capture corrected by the factor 1.18 for neutron loss.

The calculation of the incident roentgen

TABLE XI

FISSION-BURST DOSE EXPRESSED AS INCIDENT GAMMA RAYS AND SOFT ROENTGEN RAYS

Case	Incident 80 Kev. Roentgen Ray Roentgens	Incident Gamma Ray Roentgens*	Hands	
			Right Very Soft Roentgen Ray Roentgens	Left Very Soft Roentgen Ray Roentgens
1	840	487.0	20,000–40,000	5,000–15,000
2	45	3.2		
3	2036	156.0	3,000–10,000	15,000–30,000
4	407	41.0		400–600
6	210	19.0		
7	100	10.0		
8	53	6.2		
9	43	4.0		
10	30	2.6		

* 1 mev. gamma rays having linear absorption coefficient of $\mu = 0.3$ cm.$^{-1}$.

ray dose is based upon Mayneord's[10] tables and correction formulas. The correction for source-to-body distance is given in column 2, Table IX; for Case 3 it is 0.83. The average body dose/incident r is determined by Case 3's torso thickness of 20 cm. and is 0.362 r (column 4, Table IX) for plane-wave radiation. Corrected by 0.83, the average dose becomes 0.30 r/incident r (column 6).

This is the average body dose/r which Case 3 would have received at a distance of 50 cm. from an 80 kev. roentgen ray source. The 122.2 rep multiplied by the RBE factor of 5 make a total of 611 gamma-equivalent r of average body dose which have to be delivered by the roentgen rays. Thus, the incident roentgen-ray dose is 611/0.3, or 2,030 r, which is given in Table XI, column 1. Basically the calculation assumes that the critical assembly approximated a point source resembling the focal spot of a roentgen tube.

IX. BLUE-GLOW DOSE RATES

In the critical-assembly accidents of August 21, 1945, and May 21, 1946, there was reported a blue glow immediately surrounding the assembly. This blue glow had been reported earlier (June 4, 1945) when another assembly had momentarily been out of control. The operators of their respective assemblies, Cases 1 and 3, had their hands and arms in this glow. As the clinical record[4] indicates, these body members sustained extremely high radiation doses. For an estimate of the doses, it is necessary to deduce the order of magnitude of energy flux which will produce the blue glow in air.

It turns out that a glow in air due to ionizing radiations can be seen under varying degrees of background lighting: the lower the background the less intensity required to make a glow visible. The critical assemblies mentioned above produced a glow under widely differing circumstances. The first one was seen against the dark walls of a large steel tank containing the assembly. The incandescent lighting and daylight through remote windows were not bright. The second assembly glow was seen against dark blocks of shielding with incandescent light about 8 feet overhead (at 11:00 P.M.). Case 2 was reading a newspaper with his back toward the assembly. He was aware of a bright flash when this assembly went critical.

The third assembly was seen to glow in a well-lighted laboratory near large windows through which the bright afternoon (3:20 P.M.) sun shone directly. Three of the personnel present were positive in their statements of having seen a blue glow which seemed to have extended 20 cm. beyond

the critical assembly.* In view of the fact that a blue glow was seen in this brightly lighted laboratory, it is believable that Case 2 should have been aware of a bright flash on his newspaper in the night accident. Yet, it must be remembered that there were considerable differences in material surrounding the two assemblies. The afternoon assembly had relatively little material in the way of heavy metal around it as compared to the night assembly.

Various facts pertaining to blue-glow phenomena due to ionizing radiations are presented in the following discussion. A 20 microampere beam of 5 mev. deuterons can be seen readily in a darkened room. From the range-energy relation and assuming a 1 cm.^{2} beam cross section, a rough estimate is that an absorption of 2.8×10^{19} ev./cc./sec. occurs in the air at the point where the deuterons have 5 mev. energy.

Experiments with high-intensity radioactive sources showed that a blue glow easily seen in a dark room corresponded to the absorption of 4.4×10^{15} ev./cc./sec. in air. Using these data and extrapolating to those of the laboratory in which the afternoon sun gave background illumination (a factor of 10^4 in light intensity is assumed), the figure of 4.4×10^{15} ev./cc./sec. is to be raised to 10^{19}, which is comparable in magnitude to the figure of 2.9×10^{19} ev./cc./sec. for the deuteron beam. The roentgen rate is 6×10^7 r/cc./sec. for the blue glow seen in a well-lighted room.

The dose components in the blue glow comprise soft roentgen rays and electrons as well as the effects of ionization due to the primary fission radiations such as fast and slow neutrons, gamma rays, and recoil particles. In the above computation of dose rate it is assumed that the mechanism of blue-glow formation by deuterons and alpha particles is essentially the same as that for the radiations coming from the critical assembly.

The blue glow in which the hands of Cases 1 and 3 were immersed for fractions of a second produced an exposure at a high rate and of soft radiations. From the accounts given by the individuals, it is estimated that Case 1 sustained about 20,000 to 40,000 r on the right hand and 5,000 to 15,000 r on the left hand. Case 3 received 3,000 to 10,000 r on the right hand and 15,000 to 30,000 r on the left hand. Case 4 had his left hand extended toward the assembly and probably sustained 400 to 600 r on it.

* Five individuals who were near the blue g'ow in three different assemblies reported a tingling, salty sensation about their tongues while the glow was visible to them.

X. DISCUSSION OF DOSES

There are four different ways in which the whole-body dose can be designated, no one of which has been accepted as the preferred way. This may be due to the meager clinical experience reported in the literature of whole-body irradiation. There may be other ways of specifying whole-body dose; the four which are pertinent to the fission-burst accidents are discussed as follows:

1. The designation of an average value of the rep absorbed throughout the body. The essential figures for this dosage are given in Table VIII, columns 1, 2, 3, and 4. The dose presented in rep or r is simple. If it is desired to account for extreme exposure on one side of the body as occurred in Cases 1 and 3, the average rep provides a convenient reference point from which the dose can be raised or lowered as the case may require. The doses in Table VIII are corrected for estimated slow-neutron-diffusion loss and for fast-neutron scatter from the body.

2. The second specification of whole-body dose is an extension of the average body rep to total whole-body dose as in the concept of integral dose. The total rep is the product of the average body rep times the body mass as in Table V, column 5, where the rep have not yet been corrected for slow-neutron loss and fast-neutron scatter which amounts to an additional 30 per cent for the H^1-recoil dose and 18 per cent for the N^{14}-capture dose. The integral dose in terms of gamma equivalent dose is summarized in Table X. The conversion of heavy-particle rep to gamma rep was made using the RBE factor of 5.

For practical purposes the gm.r in this report is taken as 93.1 ergs/gm. of tissue. The assumption is that all the various gamma rays give 1 rep/roentgen in any tissue of the body (see Mayneord,[11] p. 144, for units and discussion).

3. The expression of body doses in terms of the incident dose rather than an average dose throughout the tissue. This statement of dose stems from radiotherapeutic experience in which the clinical reaction is measured by incident dose in well-defined radiation applicators. The doses in this form are stated for two different types of radiation. In Table VIII, column 6, the incident gamma radiation from the assembly is given which would produce the average gamma body dose in column 5. This in turn is the sum of the four dose components shown in columns 1, 2, 3, and 4.

It is recognized that the gamma equivalent of all the doses is artificial in that the heavy-particle component was concentrated toward one side of the body while the gamma doses were uniformly distributed. A single figure such as that shown in Table VIII, column 6, is not readily interpreted to show the physical distribution of dose.

The difficulty in the single gamma-equivalent dose is avoided by describing the exposure in terms of two different radiations. The heavy-particle dose is given in terms of incident 80 kev. roentgen rays and the gamma component in terms of an incident gamma dose as in Table XI.

The recourse to a soft roentgen ray dose is somewhat arbitary. Yet it gives a better picture in terms of a known therapeutic radiation. Its use is justified to some extent by the fact that fission bursts will invariably involve scattering materials around the source of neutrons. The degraded neutron-fission spectrum will be the rule rather than the exception. Further data may show the properties of a voltage other than 80 kev. to be more appropriate for the heavy-particle component of dose. The actual roentgen ray voltage is irrelevant except insofar as it provides a working guide for the radiologist in terms of a familiar tool.

4. The fourth index of whole-body dose is peculiar to neutron exposures. It is the specific activity of the Na^{24} in blood serum (Table II, column 1) as has already been pointed out in the clinical report on the 9 cases.[4] The P^{32} activities have yet to be studied in the human being. The complexities apparent in its elimination preclude its being useful at this time. In the earlier report on the accidents[5] it was noted that the appearance of measurable P^{32} in blood serum indicated a lethal dose.

The use of induced activity as an index of radiation injury requires, however, that the presence of fast neutrons be ascertained from other evidence, since readily accessible induced activities give no clue to the energy spectrum of fast neutrons. This problem has been discussed in Section VI in connection with the estimate of dose by the gamma rays coming from Na^{24} in the body.

The earlier doses published in the clinical report[4] (p. 292) are lower than those given here. The integral doses in Table I[4] (p. 292) correspond to those in Table X above except that in Table X the RBE factor of 5 has been applied in the heavy-particle dose. Table II[4] (p. 293), giving the incident doses in terms of soft roentgen rays and gamma rays, corresponds to Table XI above.

In this report the dose of delayed gamma radiation of Case 1 has been increased on the basis of the high intensity of delayed gamma rays known to have existed and the estimated length of time spent near the assembly. This has been discussed in Section IV.

The doses in this paper are higher than those in the clinical report for several reasons, although they are not significantly higher except for the special circumstance of Case 1 discussed above. The capture cross sections for slow neutrons in Na^{23} and in H^1 are taken as 0.45 and 0.31 barns respectively, whereas in the 1948 report these were 0.693 and 0.25 barns. The correction

for slow-neutron-diffusion loss and fast-neutron scatter from the body was not made in the earlier dose calculations. In this report the source-to-body distance factor has been taken into account, whereas in the earlier report the doses were given in terms of plane-wave incident radiation. While the distance factor entails the undesirable assumption that the critical assembly was a point source, the results seem less artificial than the use of the plane-wave concept. The distance factor raises the incident dose.

The heavy-particle integral dose in column 1, Table x, and its equivalent soft roentgen ray incident dose in Table xi, column 1, reduced to 77.8 per cent of their values, give the doses without the neutron-loss corrections. For example, in Case 3 the soft roentgen ray dose is 2,036 r. Reduced to 77.8 per cent, it becomes 1,585 r.

The absolute magnitude of the doses is determined by the average fast-neutron energy as discussed in Section vii above. In the first appraisals of the exposures an average energy of 0.35 mev. for the neutrons had been assumed. In the accident involving Cases 3 to 10 the material about the assembly was such as to make an estimate of 0.5 mev. seem in order.

There is the possibility that the average energy of 0.35 mev. is appropriate to the accident involving Cases 1 and 2 because of the large amounts of shielding material present. The doses in those cases might thus be considered slightly high. In the other cases it is believed that the doses due to neutron exposure are conservatively estimated and low. Certainly, if the corrections for slow-neutron loss and fast-neutron scatter are removed, the resultant doses must be near the absolute minimums possible.

The possible revision upward has already been indicated in Section vii above and involves the additional dose due to fast neutrons. The increase of 45 per cent in the heavy-particle dose by the presence of 5 per cent of neutrons having a 5 mev. average energy appears to give the order of magnitude of the upward estimates. Although this kind of estimate is consistent with the general knowledge about the fission spectrum, it has not been added to the doses reported here. Such upward revisions will have to await more detailed information about the neutron-fission spectrum.

SUMMARY

In two nuclear accidents involving uncontrolled fission reactions 9 persons were inadvertently exposed to complex ionizing radiations. Whole body doses are computed on the assumption that serum Na^{24} slow-neutron-induced activity provides a measure of fast-neutron intensity.

The selfdosage in the human body due to gamma radiation from slow neutron capture in hydrogen is computed on the basis that the serum Na^{24} activity gives a measure of slow-neutron density. Measurements of gamma ray intensities about a mock-up man filled with Na^{24} in water are described. The assay of induced Na^{24} in the sera of humans by a measurement of gamma ray intensity at the anterior diaphragm is discussed.

Slow-neutron-induced activities although measurable are found to be negligible insofar as radiation dosage is concerned.

In decreasing order of magnitude the dose components are: fast neutrons, hydrogen capture gamma rays, N^{14} captures, and prompt gamma rays.

Doses are expressed in: (a) average rep throughout the body; (b) integral dose; (c) terms of incident r delivered by 1 mev. gamma and soft roentgen rays; and (d) the serum Na^{24} induced activities.

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EDITORIAL

ROENTGENOLOGIC MANIFESTATIONS OF EMOTIONAL DISTURBANCES IN THE STOMACH

RADIOLOGISTS are, by the nature of their diagnostic work, conditioned to focus their attention on organic, *i.e.* morphologic changes. Nevertheless their observations also afford them an excellent opportunity to evaluate the functional status of the organ under investigation. This is particularly true for the stomach. As early as 1898 Cannon,[7] in his fundamental roentgenologic studies on the movements of the stomach, was able to show that in the cat, excitement, anxiety, rage or fear resulted in cessation of peristaltic waves and relaxation of the stomach.

The two dynamic phenomena which are particularly accessible to roentgenologic investigation are tonus and peristalsis of the stomach. They are closely interrelated and are governed mainly by the intrinsic autonomic plexuses in the wall of the stomach, but are also influenced by its extrinsic innervation, the vagus and sympathetic. The results of vagal or sympathetic stimulation on the stomach are not clear cut, but depend on the degree of tonus and the activity of peristaltic motion at the time of stimulation as well as on the frequency and strength of the stimulus.[3] In general the vagus increases tonus and motility of the stomach and relaxes the pyloric sphincter while the sympathetic is inhibitory to the stomach wall and excitatory to the pyloric sphincter. There is evidence of cortical and subcortical control of gastric motility. The centers in the cortex most likely act upon the hypothalamus which in turn affects the vagal and sympathetic outflow. At the present state of our knowledge we have to assume with Youmans[30] that there are mechanisms by means of which influences from the higher centers of the brain may be brought to bear upon gastrointestinal motility. Normally an equilibrium seems to exist between excitatory and inhibitory influences which maintains an intermediate level of gastric tonus and motility. It can be conceived that in certain emotional states one or the other of the central governing mechanisms may become hyperactive, resulting in abnormal motor functions of the gastrointestinal tract. Which pattern of response is elicited may depend not only on the type of emotional stimulus, but also on the personality make-up of the individual.

It is surprising that in spite of the acute interest in functional gastrointestinal disturbances, the radiologist is only rarely called upon, beyond the request for exclusion of organic lesions, to give his observations on gastrointestinal physiology to the psychosomatically oriented clinician.

While the roentgenologic approach is of little assistance in the demonstration of secretory phenomena, the reverse is true with respect to motor physiology. To quote Bockus, "The roentgen method offers the only consistently reliable reproducible technique for the study of motor activity of the digestive tract."[4] Nevertheless the amount of available roentgenologic data on gastric psychodynamics is limited and the potentialities of the roentgenologic method have certainly not been exhausted.

So far the major portion of evidence on the interrelationship of gastric function and emotional processes has been gathered by methods outside the realm of radiology. Wolf and Wolff[29] in their now classic experiments on their subject "Tom," a man with a gastric fistula, demonstrated by direct observation of the gastric mucosa, by aspiration of stomach contents, and by recording of pressure changes in an in-

dwelling balloon, that emotional states were reflected in the secretory and motor behavior of the organ. Anxiety, anger, hostility, and resentment led to vascular engorgement, hypersecretion, and hypermotility. On the other hand, fear, dejection, and a general reaction of withdrawal were associated with a depression of these functions. In later studies Wolf[27] was able to show that as the face may blanch or blush in varying situations of stress, so may the stomach become either hypoactive or hyperactive, depending on the situation and the individual's reaction to the situation. Margolin[16] has carried out simultaneous psychoanalytic sessions and physiologic studies in a patient with gastric fistula and has correlated the patient's unconscious and conscious emotional disturbances with demonstrable functional changes in the stomach.

All of these important investigations were made without the benefit of the roentgenologic method, yet it must not be overlooked that the introduction into the stomach of a foreign body such as an inflated balloon, the manipulations of the investigator on the gastric fistula, and the over-all reaction of the subject to the environmental setting of the experiment, all contribute their share to the physiologic outcome of the experiment. As Wittkower and Cleghorn[26] have stated recently, there is no doubt that the psychologic responses of the subject mobilized by the experimental procedure can significantly influence the physiologic result. By comparison, the roentgenologic method, if properly executed, recommends itself by its less aggressive approach to the subject. Yet this holds true only if the locale of the roentgenologic examination is consciously controlled to avoid stresses on the subject under study. Kirklin[15] has called attention to the fact that the slightest apprehension of the patient, caused by his surroundings or by unskillful handling of the patient on the part of the physician or technical personnel, will find its roentgenologic expression in increased tonus of the stomach, in gastric spasm, and in elevation and delayed emptying of the organ. Attendants are required to speak in low and pleasant tones, to shun any appearance of haste or excitement, to keep their hands off patients and to treat them with all respect and dignity. Brief but friendly conversation with the patient during the examination will promote his serenity.[15]

Interesting in this respect also are the fluoroscopic observations by Ruggles.[19] A patient with a rather atonic stomach became enraged when her roentgenologic examination was interrupted to permit the examination of another patient to be completed. When she was re-studied, her stomach had risen, contracted to half its former size and showed rapid, vigorous peristalsis. When her anger subsided, the stomach returned to its previous state. The opposite effect of dropping of the stomach with relaxation of the gastric wall has been noted by Barclay,[2] who observed it in conjunction with startling of the patient by acoustic stimuli, with apprehension on first examination, and preceding fainting. When quiet was re-established, apprehension relieved, or the patient restored to consciousness, the stomach assumed its normal appearance.

These and other incidental observations, which can be readily supplemented by any experienced radiologist, represent in themselves important data in the field of psychophysiology, but even more revealing are investigations which intentionally introduce situational stresses concomitant with roentgenologic study of the motor activity of the stomach.

The anatomist Todd[23] interviewed freshman medical students with questionable scholastic record during fluoroscopy by discussing their class standing with them. He noted definite gastric response resulting either in rising or lowering of gastric position. Later he found that the change in gastric position generally was paralleled by the reaction of the student to his critical class standing. Those whose stomachs rose applied themselves with greater effort and

were able to enter the sophomore class, while those who responded with a drop generally failed to obtain promotion.

Very interesting are early roentgenologic observations on gastric motility by Heyer,[12] who employed hypnosis simultaneously with fluoroscopy on patients with dyspeptic complaints. Using a first roentgenologic examination in the waking state as a control, he applied hypnosis on re-examination, inducing disgust with the meal. This resulted in cessation of peristalsis and complete loss of tonus. Emptying of the stomach consequently stopped. When, on the other hand, pleasurable associations with the barium meal were established, the stomach returned to its previous tonus, peristaltic waves increased, and emptying time was accelerated. Patients under emotional stress whose stomachs had been atonic were able, under suggestive therapy, to overcome the atony. As the author readily admits, the rising of the stomach can, in part, be ascribed to increased tonus of the anterior abdominal wall.

So far the experimental studies mentioned have utilized situational stresses that were derived mainly from the life histories of the individuals under study. Jungmann and Venning,[13,14] on the other hand, explored the stomach fluroscopically in an artificially stressful situation by exposing the subject to a sudden threat of a needle injection or an unexpected loud auditory stimulus. In about half of the cases subjected to the experiment, changes in peristalsis occurred during the period of stress, consisting of either cessation of peristalsis or decrease in intensity of the waves.

In a recent statistical study Mangelsdorff[18] investigated roentgenologically the gastric tonus of more than 1,000 German subjects with a negative clinical history and otherwise normal roentgenologic gastric findings. He observed a general tendency to hypertonus in the critical postwar period, occurring in 62 per cent of all individuals under study in the early postwar years (1947–1948). It decreased in frequency to 34 per cent, as times became better and more tranquil (1949–1950). The author also noted a significant rise in tonus in spring and fall, which interestingly enough coincides with the seasonal peak in the occurrence of peptic ulcer.

In separate papers, based on work done with the same group of patients, Brummer[5] and Wegelius[24] have called attention to the close correlation that exists between the presence of gastric distress and roentgenologically demonstrable motility disturbances consisting of combinations of various degrees of increased tonus and increased peristalsis. Ninety per cent of the patients showed these functional abnormalities in gastric distress, whereas the corresponding figure in healthy people is only 10 to 15 per cent. The authors point out that the presence of motility disturbances does not necessarily give rise to dyspeptic symptoms, but they believe that functional abnormalities are the immediate cause of the distress and assign to psychic factors and emotional states an important share in the causation of these disturbances.

Well known is the close association of psychologic trauma, clinical symptoms and roentgenologically demonstrable circumscribed spasm, particularly of the cardia.[17] Leaving aside details of the physiologic mechanism underlying cardiospasm, it is certain that the degree of obstruction at the cardia can be modified predictably by variations in the emotional state of the individual. Such modifications can be brought about under fluoroscopy by a discussion of emotionally charged topics. Even in normal subjects cardiospasm occasionally can be induced in response to general stress, including the threat of an oral examination in medical students.[28] Spasms in other parts of the stomach may likewise be of psychogenic origin and occur in hysteria, in anxiety states, and endogenous psychoses.[20] They may be total, involving the whole organ,[9] or regional, and are interpreted as caused by reflex stimulation of the parasympathetic system.

Whether the delay in gastric emptying in emotionally unstable subjects is due to

pylorospasm or temporary peristaltic inactivity and hypotonicity of the stomach is an open question. The latter seems more likely,[1,4,22] particularly in view of the readily demonstrable stimulating effect that the discussion of pleasure-yielding food produces on gastric motility.[8] Buckstein[6] notes that even a six-hour residue of as much as one fourth of the barium may be purely functional. Re-examination of the subject at a time, when his anxiety has been relieved, may show the emptying time of the stomach considerably closer to the normal.

Buckstein[6] and, previously, also Wittkower[25] noted that the mucosal folds may become prominent and tortuous as the result of affective influences simulating the picture of true gastritis. Schinz and his co-authors[20] speak in this connection of a hypertonicity of the muscularis mucosae governed by the autonomic Meissner plexus.

Brief mention, finally, should be made of important studies by Henry[10,11] on the relation of roentgenologically observable gastrointestinal motor functions to personality disorders of the psychotic type. He observed that the function of the stomach varies according to the character of mental illness. In the manic phase of manic-depressive psychoses the position of the viscera is from 1 to 2 inches higher than in the depressive phase. Hypomanic patients present a marked increase in visceral tonus and motility, while in manic patients visceral function has passed the limit of acceleration and begins to be retarded. Depressed patients present a pronounced decrease in visceral tonus and motility. Henry also found definite changes in gastrointestinal motor function in schizophrenic patients, particularly of the more acute type.

Throughout the relatively short history of the roentgenologic approach to the study of the gastrointestinal tract and particularly of the stomach, interest in functional changes has wavered.[21] In the early days of the use of the barium meal the observation of functional and organic alterations was regarded as of equal importance. This was the era of indirect evidence of organic lesions, which was properly replaced by the attempt at direct demonstration of organic pathology. It led to a regrettable disregard for observable alterations in gastric dynamics. These changes, though not pathognomonic, nevertheless represent important indicators of the psychosomatic status of the individual that should find a proper place in the functional assessment of the digestive system. Fruitful results can be expected if the radiologist, with his modern methods of roentgen kymography and roentgen cinematography (in addition to standard procedures), will participate to a greater extent in the work of the psychiatrist, the psychosomatically oriented gastroenterologist, and the physiologist interested in visceral psychodynamics.

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SOCIETY PROCEEDINGS AND NEWS ITEMS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. Barton R. Young, Germantown Hospital, Philadelphia 44, Pa. Annual meeting, Shoreham Hotel, Washington, D. C., Oct. 1–4, 1957.

American Radium Society
Secretary, Dr. Theodore R. Miller, New York, N. Y. Annual meeting: Chateau Frontenac, Quebec, Canada, May 30–June 1, 1957.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 713 E. Genesee St., Syracuse 2, N. Y. Annual meeting: To be announced.

American College of Radiology
Executive Secretary, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Illinois. Annual meeting: Febr. 8, 1957, Drake Hotel, Chicago, Illinois.

Section on Radiology, American Medical Association
Secretary, Dr. T. Leucutia, Harper Hospital, Detroit 1, Mich. Annual Meeting, June 3–7, 1957, New York, N. Y.

Eighth International Congress of Radiology
Secretary General, Dr. José Noriega, Calle del Oro 15, Mexico 7, D. F.

Alabama Radiological Society
Secretary, Dr. J. A. Meadows, Jr., Medical Arts Bldg., Birmingham 5, Ala. Meets time and place Alabama State Medical Association.

American Nuclear Society
Dr. L. D. P. King, Los Alamos Scientific Laboratory, Post Office Box 1663, Los Alamos, N.M.

Arizona Radiological Society
Secretary, Dr. J. J. Riordan, 550 W. Thomas Rd., Phoenix, Ariz. Two regular meetings a year. The annual meeting at time and place of State Medical Association and interim meeting six months later.

Arkansas Radiological Society
Dr. E. A. Mendelsohn, Holt-Krock Clinic, Fort Smith, Ark. Meets every three months and also at time and place of State Medical Association.

Association of University Radiologists
Secretary, Dr. David M. Gould, University of Arkansas Medical Center, Little Rock, Ark. Annual meeting, May, 1957 at the University of Minnesota.

Atlanta Radiological Society
Secretary, Dr. Charles M. Silverstein, 3254 Peachtree St., N.E., Atlanta, Ga. Meets monthly, except during three summer months, on second Friday evening.

Blockly Radiological Society
Secretary, Dr. Samuel Finkelman.

Brooklyn Radiological Society
Secretary, Dr. Harold N. Schwinger, 62 8th Ave., Brooklyn, N. Y. Meets first Thursday of each month October through May.

Buffalo Radiological Society
Secretary, Dr. Charles Bernstein, 685 Delaware Ave., Buffalo, N. Y. Meets second Monday evening each month, October to May inclusive.

Central New York Roentgen Ray Society
Secretary, Dr. Dwight V. Needham, 608 E. Genesee St., Syracuse, N. Y. Meets January, May, November.

Central Ohio Radiological Society
Secretary, Dr. Arthur R. Cohen, 41 S. Grant Ave., Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, November, January, March and May at Fort Hayes Hotel, Columbus, Ohio.

Central Society of Nuclear Medicine
Secretary, Dr. Robert S. Landauer, Radiation Center Building, 1903 West Harrison St., Chicago 12, Ill.

Chicago Roentgen Society
Secretary, Dr. Arthur S. J. Petersen, 11406 Parnell Ave., Chicago 28, Ill. Meets second Thursday of each month, October to April inclusive at the Sheraton Hotel.

Cleveland Radiological Society
Secretary, Dr. A. S. Tucker, 2065 Adelbert Rd., Cleveland 6, Ohio. Meetings at 7:00 P.M. on fourth Monday of each month from October to April at Tudor Arms Hotel.

Colorado Radiological Society
Secretary, Dr. Lorenz R. Wurtzebach, 601 Nineteenth Ave., Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

Connecticut Valley Radiologic Society
Secretary, Dr. T. J. Crowe, 53 Center St., Northampton, Mass. Meets second Friday October and April.

Dallas-Fort Worth Radiological Club
Secretary, Dr. A. H. Keene, 3707 Gaston Ave., Dallas, Texas. Meets monthly, third Monday, at Greater Fort Worth International Airport at 6:30 P.M.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. Joseph O. Reed, Jr., Harper Hospital, Detroit, Mich. Meets monthly first Thursday, Oct. through May, at David Whitney House, 4421 Woodward Ave., at 6:30 P.M.

East Bay Roentgen Society
Secretary, Dr. Dan Tucker, 434 30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

Florida Radiological Society
Secretary, Dr. C. Robert DeArmas 135 Broadway, Daytona Beach, Fla. Meets twice annually, in the spring with the annual State Society meeting, and in the fall.

Georgia Radiological Society
Secretary, Dr. B. H. Malone, 1406 Reynolds St., Brunswick, Ga.

Greater Miami Radiological Society
Secretary, Dr. A. S. Capi, 300 N. 20th Ave., Hollywood, Fla. Meets monthly third Wednesday at 8 P.M. at Mercy Hospital, Miami, Fla.

Greater St. Louis Radiological Society
Secretary, Dr. Sam J. Merenda.

Houston Radiological Society
Secretary, Dr. Leslie L. Lemak, 616 Medical Arts Bldg., Houston 2, Texas. Meets fourth Monday each month, Meeting Room, Doctors' Club of Houston.

Idaho State Radiological Society
Secretary, Dr. Alfred M. Stone. St. Lukes Hospital, Boise, Idaho. Meets annually at same time as Idaho State Medical Association.

Illinois Radiological Society
Secretary, Dr. Stephen L. Casper, 1101 Maine St., Quincy, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. C. A. Stayton, 313 Hume-Mansur Bldg., Indianapolis 4. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

Iowa Radiological Society
Secretary, Dr. James T. McMillan, 1104 Bankers Trust Bldg., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

* Secretaries of societies are requested to send timely information promptly to the Editor.

KANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. A. M. Cherner, Hays, Kansas. Meets in spring with State Medical Society, and in winter on call.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. David Shapiro, 217 East Chestnut St., Louisville 2, Ky. Meets monthly on second Friday at Seelbach Hotel, Louisville.

KINGS COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Ernest I. Melton, 2187 Ocean Ave., Brooklyn 19, N.Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

LOS ANGELES RADIOLOGICAL SOCIETY
Secretary, Dr. Lewis J. Peha, 405 North Bedford Drive, Beverly Hills, Calif. Meets second Wednesday each month at Los Angeles County Medical Association Building, Los Angeles.

MAINE RADIOLOGICAL SOCIETY
Secretary, Dr. Irving L. Selvago, Jr., Maine Medical Center, 22 Arsenal St., Portland, Maine. Meets in June, September, December and March.

MARYLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Nathan B. Hyman, 1805 Eutaw Place, Baltimore, Md.

MEMPHIS ROENTGEN SOCIETY
Secretary, Dr. Dan C. Gary, 36 South Bellevue, Memphis 4, Tenn. Meets first Monday of each month at John Gaston Hospital.

MIAMI VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. G. A. Nicoll, 134 Apple St., Dayton, Ohio. Meets second Friday of fall and winter months.

MID-HUDSON RADIOLOGICAL SOCIETY
Secretary, Dr. J. W. Walton, Horton Memorial Hospital, Middletown, N. Y. Meets 8:30 P.M., fourth Tuesday each month, September to May.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. R. W. Bryne, 3321 N. Maryland Ave., Milwaukee 1, Wis. Meets monthly on fourth Monday at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. O. J. Baggenstoss, 1953 Medical Arts Bldg., Minneapolis, Minn. Meets three times annually, once at time of State Med. Assn., and in spring and fall.

MISSISSIPPI RADIOLOGICAL SOCIETY
Secretary, Dr. James M. Packer, 621 High St., Jackson, Miss. Meets third Thursday of each month at Hotel Edwards, Jackson, at 6:00 P.M.

MONTANA RADIOLOGICAL SOCIETY
Secretary, Dr. John Stewart, 1105 Frederick Lane, Billings, Montana. Meets at least once a year.

NASSAU RADIOLOGICAL SOCIETY
Secretary, Dr. Jerome Zwanger, 126 Hicksville Rd., Massapequa, L. I., N. Y. Meets second Tuesday of the month in February, April, June, October and December.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. J. F. Kelly, Jr., 816 Medical Arts Bldg., Omaha, Neb. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Raymond A. Dillon, 24 Wedgemere Ave., Winchester, Mass. Meets third Friday of each month, October through May, at Hotel Commander, Cambridge, Mass.

NEW HAMPSHIRE ROENTGEN RAY SOCIETY
Secretary, Dr. A. C. Johnston, Elliott Community Hospital, Keene, N. H. Meets four to six times yearly.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. John A. Evans, 525 East 68th St., New York 21, N. Y. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

NORTH CAROLINA RADIOLOGICAL SOCIETY
Secretary, Dr. William H. Sprunt, North Carolina Memorial Hospital, Chapel Hill, N. C. Meets in May and October.

NORTH DAKOTA RADIOLOGICAL SOCIETY
Secretary, Dr. Marianne Wallis, St. Joseph's Hospital, Minot, N. D. Meetings by announcement.

NORTH FLORIDA RADIOLOGICAL SOCIETY
Secretary, Dr. Marvin Harlan Johnston, Five Points Medical Center, Jacksonville, Fla. Meets quarterly in March, June, September and December.

NORTHEASTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Irving Van Woert, Jr., Albany Hospital, Albany, N. Y. Meets in Albany area on second Wednesdays of October, November, March and April.

NORTHERN CALIFORNIA RADIOLOGICAL SOCIETY
Secretary, Dr. H. B. Stewart, 2920 Capital Ave., Sacramento, Calif. Meets at dinner last Monday of each month, September to June.

OHIO STATE RADIOLOGICAL SOCIETY
Secretary, Dr. John R. Hannan, 10515 Carnegie Ave., Cleveland 6, Ohio. Annual meeting, Dayton, May, 1957, to be announced.

OKLAHOMA STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Sol Wilner, St. John's Hospital, Tulsa, Okla. Three regular meetings annually.

OREGON RADIOLOGICAL SOCIETY
Secretary, Dr. N. L. Bline, 210 Jackson Tower, Portland 5, Ore. Meets monthly from October to June on the second Wednesday of each month at 8:00 P.M. at the University Club.

ORLEANS PARISH RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets first Tuesday of each month.

PACIFIC NORTHWEST RADIOLOGICAL SOCIETY
Secretary, Dr. J. R. Raines, 214 Medical-Dental Bldg., Portland 5, Oregon. Meets annually in May.

PACIFIC ROENTGEN SOCIETY
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

PENNSYLVANIA RADIOLOGICAL SOCIETY
Secretary, Dr. Walter P. Bitner, 234 State Street, Harrisburg, Pa.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. Roderick L. Tondreau, 3400 Spruce St., Philadelphia, Pa. Meets first Thursday of each month, at 5 P.M., from October to May in Thompson Hall, College of Physicians.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. N. B. Tannehill, 601 Jenkins Bldg., Pittsburgh 22, Pa. Meets second Wednesday of month, October through June at Hotel Roosevelt.

RADIOLOGICAL SECTION, BALTIMORE MEDICAL SOCIETY
Secretary, Dr. Paul W. Roman, 1810 Eutaw Place, Baltimore 17, Md. Meets third Tuesday each month, September to May, inclusive.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

RADIOLOGICAL SOCIETY OF GREATER CINCINNATI
Secretary, Dr. Morris M. Garrett, 630 Scott St., Covington, Ky. Meets monthly from September to May on first Monday of each month at 7:30 P.M. at the Cincinnati General Hospital.

RADIOLOGICAL SOCIETY OF HAWAII
Secretary, Dr. Richard D. Moore, St. Francis Hospital, Honolulu 17, T. H. Meets third Monday evening of each month at designated hospitals.

RADIOLOGICAL SOCIETY OF GREATER KANSAS CITY
Secretary, Dr. E. H. Stratemeier, Jr., 1010 Rialto Building, Kansas City 6, Mo. Meets last Friday of each month.

RADIOLOGICAL SOCIETY OF KANSAS CITY
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

RADIOLOGICAL SOCIETY OF LOUISIANA
Secretary, Dr. W. S. Neal, 602 Pere Marquette Bldg., New Orleans, La. Meets annually during Louisiana State Medical Society Meeting.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. G. G. Green, 601 Grand Ave., Asbury Park, N. J. Meets at Atlantic City at time of State Medical Society and in November in Newark, N. J.

RADIOLOGICAL SOCIETY OF SOUTHERN CALIFORNIA
Secretary, Dr. Robert B. Engle, 2632 E. Washington St., Pasadena, Calif.

Richmond County Radiological Society
Secretary Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Henry H. Forsyth, Jr., 40 Meigs St., Rochester, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. John H. Freed, 4200 East Ninth St., Denver 20, Colo.

St. Louis Society of Radiologists
Secretary, Dr. Edwin C. Ernst, Jr., 3720 Washington Ave., St. Louis 8, Mo. Meets fourth Wednesday each month, except June, July, Aug. and Sept.

San Antonio-Military Radiological Society
Secretary, Dr. Hugo F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Texas. Meets Brooke Army Medical Center Officers Mess second Wednesday of each month.

San Diego Radiological Society
Secretary, Dr. J. C. Asher, 2001 Fourth Ave., San Diego 1, Calif. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary, Dr. Irma Smith, 450 Sutter St., San Francisco 18, Calif. Meets quarterly at Grison's Steak House.

Section on Radiology, California Medical Association
Secretary, Dr. A. R. Wilson, 540 North Central Ave., Glendale 3, Calif.

Section on Radiology, Connecticut State Medical Society
Secretary, Dr. J. Burbank, Meriden Hospital, Meriden, Conn. Meetings are held bi-monthly.

Section on Radiology, Medical Society of the District of Columbia
Secretary, Dr. Melvin O. Goodman, 1930 K St., N.W., Washington 6, D. C. Meets Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. George E. Irwin, Jr., 703 N. East St., Bloomington, Ill.

Shreveport Radiological Club
Secretary, W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M. September to May inclusive.

Society of Nuclear Medicine
Secretary, Dr. Robert G. Moffat, B. C. Cancer Institute, 2656 Heather Street, Vancouver 9, Canada. Annual meeting to be announced.

South Bay Radiological Society
Secretary, Dr. T. N. Foster, 630 E. Santa Clara St., San Jose, Calif. Meets second Wednesday of each month.

South Carolina Radiological Society
Secretary, Dr. S. W. Lippincott, 103 Rutledge Ave., Charleston, S. C. One meeting with South Carolina Medical Association in May, others arranged by President.

Southwestern Radiological Society
Secretary, Dr. Gordon Black, Suite 2-A, El Paso Medical Center, 1501 Arizona, El Paso, Texas. Meets second Tuesday of each month.

Tennessee Radiological Society
Secretary, Dr. G. K. Henshall, 311 Medical Arts Bldg., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. J. E. Miller, Baylor Hospital, Dallas, Texas. Next meeting, Corpus Christi, Texas, Jan. 18–19, 1957.

Tri-State Radiological Society
Secretary, Dr. Robert E. Beck, 600 Mary St., Evansville, Ind. Meets last Wednesday of Oct., Jan., March and May, 8:00 P.M. at Elks' Club in Evansville.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets first and third Thursdays 4:00 to 5:00 P.M., September to May, Service Memorial Institute.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society
Secretary, Dr. Angus K. Wilson, 343 S. Main St., Salt Lake City 1, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Virginia Radiological Society
Secretary, Dr. P. B. Parsons, Norfolk General Hospital, Norfolk, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Eva L. Gilbertson, 1317 Marion St., Seattle 4, Wash. Meets fourth Monday of each month from September through May at 610 Pine St., Seattle.

West Virginia Radiological Society
Secretary, Dr. W. Paul Elkin, 515 Medical Arts Bldg., Charleston, W. Va. Meets concurrently with annual meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester Radiological Society
Secretary, Dr. M. G. Priestman, New Rochelle Hospital, New Rochelle, N. Y. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society
Secretary, Dr. Farrell F. Golden, 5221 Tonyawatha Trail, Madison, Wis. Special meetings held during year; annual meeting each spring at Madison, Wis.

X-Ray Study Club of San Francisco
Secretary, Dr. J. T. English, 2000 Van Ness Ave., San Francisco 9, Calif. Meets monthly, third Thursday at 7:45 P.M., Lane Hall, Stanford University Hospital.

Cuba, Mexico, Puerto Rico and Central America

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

Sociedad Costarricense de Radiologia
Secretary, Dr. James Fernández Carballo, Apartado VIII, San Jose, Costa Rica.

Sociedad Mexicana de Radiología, A. C.
Calle del Oro No. 15, Mexico 7, D. F.
Secretary General, Dr. Guillermo Santín. Meets first Monday of each month.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Diaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sanchez, Apartado No. 86, Panama, R. de P. Meets monthly in a department of radiology of a local hospital, chosen at preceding meeting.

British Empire

Association of Radiologists of the Province of Quebec
Secretary, Dr. Isadore Sedlezky, 3755 Cote St. Catherine Rd., Montreal, Que. Meets four times a year.

British Institute of Radiology Incorporated with the Röntgen Society
Ordinary meeting, on the Thursday preceding the third Friday, October to May at 8:15 P.M.
Medical Members' meeting, on third Friday in each month at 5:00 P.M., 32 Welbeck St., London, W 1

Faculty of Radiologists
Honorary Secretary, Dr. R. A. Kemp Harper, 45, Lincoln's Inn Fields, London, W.C.2, England.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London.

Canadian Association of Radiologists
Honorary Secretary-Treasurer, Dr. Guillaume Gill, Suite 305, 1555 Summerhill Avenue, Montreal 25, Que. Meetings January and June.

SECTION OF RADIOLOGY, CANADIAN MEDICAL ASSOCIATION
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

SOCIÉTÉ CANADIENNE-FRANÇAISE D'ELECTRO-RADIOLOGIE MÉDICALES
General Secretary, Dr. Ls Ivan Vallée, 1058 rue St-Denis, Montreal 18, Canada. Meets third Saturday each month.

COLLEGE OF RADIOLOGISTS (AUSTRALIA AND NEW ZEALAND)
Honorary Secretary, Dr. Alan R. Colwell, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W.

SOUTH AMERICA

ASOCIACION ARGENTINA DE RADIOLOGIA
Secretary, Dr. Lidio G. Mosca, Avda. Gral. Paz 151 Córdoba, Argentina. Meetings held monthly.

SOCIEDAD ARGENTINA DE RADIOLOGIA, JUNTA CENTRAL, BUENOS AIRES
Secretary, Dr. Oscar F. Noguera, Santa Fe 1171, Buenos Aires. Meetings are held monthly.

SOCIEDAD ARGENTINA DE RADIOLOGIA, FILIAL DEL LITORAL, SEDE, ROSARIO
President, Dr. Francisco P. Cifarelli. Meets monthly second Wednesday, 19 ha 663 Italia St., Rosario, Santa Fe.

SOCIEDAD ARGENTINA DE RADIOLOGIA, FILIAL DEL SUR, SEDE, LA PLATA
President, Prof. Dr. Jose P. Uslenghi, La Plata. Meets monthly.

SOCIEDAD ARGENTINA DE RADIOLOGIA, FILIAL DEL CENTRO SEDE, CÓRDOBA
President, Prof. Dr. David L. Caro, Corro 86, Córdoba. Meets monthly.

SOCIEDAD ARGENTINA DE RADIOLOGIA, FILIAL DEL NORTE, SEDE, TUCUMAN
President, Dr. Julio Mendez Valladares, Rivadavia 358, Tucuman. Meets monthly.

SOCIEDAD ARGENTINA DE RADIOLOGIA, FILIAL DE CUYO SEDE, MENDOZA.
President, Dr. J. A. Orfila, Hospital Central, Mendoza. Meets monthly.

SOCIEDADE BRASILEIRA DE RADIOLOGIA MEDICA
Secretary, Dr. Nicola Caminha, Av. Mem. de Sa, Rio de Janeiro, Brazil. Meets monthly, except during January, February and March.

SOCIEDADE BRASILEIRA DE RADIOTERAPIA
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644 São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

SOCIEDAD CHILENA DE RADIOLOGIA
Secretary, Dr. Armando Doberti, Merced 565, Santiago, Chile. Meets fourth Friday of each month.

SOCIEDAD COLOMBIANA DE RADIOLOGIA
Secretary, Dr. Luis E. Lozano B., Carrera 12 No. 20–69, 6° piso, Bogota, Colombia. Meets last Thursday of each month.

SOCIEDAD ECUATORIANA DE RADIOLOGIA Y FISIOTERAPIA
Secretary, Dr. Enrique Ortega Guzmán, Casilla 4719, Guayaquil, Ecuador.

SOCIEDAD PERUANA DE RADIOLOGIA
Secretary, Dr. Luis Pinillos G. Apartado 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta 218, Lima.

SOCIEDAD DE RADIOLOGIA DEL ATLANTICO
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiologia.

SOCIEDAD DE RADIOLOGÍA, CANCEROLOGÍA Y FÍSICA MÉDICA DEL URUGUAY
Secretary General, Dr. Roberto Francois, Av. Agraciada 1464, Piso 13, Montevideo, Uruguay.

SOCIEDADE DE RADIOLOGIA DE PERNAMBUCO
Secretary, Dr. Manoel Medeiros, Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

SOCIEDAD VENEZOLANA DE RADIOLOGÍA
Secretary, Dr. Otto Paz, Centro Médico, Caracas, Venezuela. Meets monthly third Friday at Colegio Médico del Distrito Federal, Caracas.

CONTINENTAL EUROPE

AUSTRIAN ROENTGEN SOCIETY
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

SOCIÉTÉ BELGE DE RADIOLOGIE
General Secretary, Dr. S. Masy, 256 Chaussée de Wavre, Heverlee-lez-Louvain, Belgium. Meets monthly, second Sunday at Maison des Médecins, Brussels.

SOCIÉTÉ FRANÇAISE D'ELECTRO-RADIOLOGIE MÉDICALE, and its branches: SOCIÉTÉ DU SUD-OUEST, DU LITTORAL MÉDITERRANÉEN, DU CENTRE ET DU LYONNAIS, DE L'OUEST, DE L'EST, ET D'ALGER ET D'AFRIQUE DU NORD
Secretary-General, Dr. A. Dariaux, 9 bis, Boulevard Rochechouart, Paris IX°, France.

CESKOSLOVENSK SPOLEČNOST PRO RËNTGENOLOGII A RADIOLOGII V PRAZE
Secretary, Dr. Roman Bláha, Praha XII, stát. nemocnice, Czechoslovakia. Meets monthly except during July August, and September. Annual general meeting.

DEUTSCHE RÖNTGEN-GESELLSCHAFT
Secretary, Dr. med. Peter Kröker, Evangelisches Krankenhaus, Huyssens Stiftung, (22a) Essen.

SOCIETA ITALIANA DI RADIOLOGIA MEDICA
Secretary, Prof. Mario Ponzio, Ospedale Mauriziano, Torino, Italy. Meets biannually.

NEDERLANDSE VERENIGING VOOR ELECTROLOGIE EN ROENTGENOLOGIE
Secretary, Dr. B. J. Rethmeier, Bornsestraat 378, Almelo Netherlands.

POLISH SOCIETY OF RADIOLOGY
Secretary, Dr. Juljusz Borejko, 59 Nowogrodzka St., Warsaw, Poland.

GDANSK SECTION, POLISH SOCIETY OF RADIOLOGY
Secretary, Dr. A. Smigielska, Akademia Lekarska, Gdansk. Meets monthly last Sunday at 10:30, X-Ray Dept., Akademia Gdansk.

WROCLAW SECTION, POLISH SOCIETY OF RADIOLOGY
Secretary, Dr. R. Marciniak, Akad. Medyczna Wroclaw, 68 Curie-Sklodowska St., Wroclaw, Poland. Meets first Sunday of each month, X-Ray Department, Akademia Medyczna Wroclaw.

SOCIETATEA ROMANA DE RADIOLOGIE SI ELECTROLOGIE
Secretary, Dr. Oscar Meller, Str. Banual Mărăcine, 30 S.I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

ALL-RUSSIAN ROENTGEN RAY ASSOCIATION, LENINGRAD
USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

SCANDINAVIAN ROENTGEN SOCIETIES
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

SOCIEDAD ESPAÑOLA DE RADIOLOGIA Y ELECTROLOGIA
Secretary, Dr. D. Aureo Gutierrez Churruca, Esparteros, No. 9, Madrid, Spain. Meets monthly in Madrid.

SCHWEIZERISCHE RÖNTGEN-GESELLSCHAFT (SOCIÉTÉ-SUISSE DE RADIOLOGIE)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

INDIA

INDIAN RADIOLOGICAL ASSOCIATION
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

The list of Meetings of Radiological Societies is published in every other issue of the JOURNAL.

ORINS PRESENTS THYROID-UPTAKE SEMINARS

The Medical Division of the Oak Ridge Institute of Nuclear Studies has announced that it will present a series of six thyroid-uptake seminars this winter and spring, to provide instruction in a standard method of calibrating the uptake of radioiodine by the thyroid. Two of the sessions will be open to invited physicians and physicists; two will be open to all other qualified physicians and physicists; one will be designed for manufacturers' representatives; and one will be open to qualified technicians.

Seminars A and B: January 14–15 and February 11–12. Limited to participants in the ORINS survey of thyroid-uptake measurement. These seminars will be devoted to lectures, demonstrations, laboratory work, and discussions of the phantom, the formula, the standard, the spectrum, and other parameters of variation.

Seminars 1 and 2: March 18–19 and April 15–16. Open to qualified physicians and physicists who have not participated in the thyroid-uptake calibration survey. Each of these seminars will accommodate a total of 30 persons, with a maximum of 15 physicists. A $25.00 fee will be charged, and the program will be the same as for Seminars A and B.

Seminar 3: May 17. This seminar will be open only to representatives of manufacturers of thyroid-uptake equipment. A maximum of 60 participants will be accepted, and a $25.00 fee will be charged. The program will be similar to that for the previous seminars, with laboratory sessions omitted.

Seminar 4: June 10–14. A total of 30 technicians employed under the direction of physicians who have attended one of the previous seminars will be accepted in this experimental seminar, which will train technical assistants for work with professional staff members. A fee of $25.00 per person will be charged.

Further information concerning any of the seminars, and application forms, may be obtained by writing Mr. William Busby, ORINS Medical Division, P. O. Box 117, Oak Ridge, Tennessee.

DR. L. ARRIETA SÁNCHEZ HONORED

At the Seventh Argentinian Congress of Radiology held in Rosario, September 27–29, 1956, the Gold Medal "Mentor de la Radiologia Americana" was conferred upon Dr. Luis Arrieta Sánchez of Panama in recognition of his great and important contribution to radiology and devoted service as Editor of *Radiología*, and for his continued efforts in introducing new techniques and modern methods in the radiology of his country.

NINTH ANNUAL MID-WINTER RADIOLOGICAL CONFERENCE

The Ninth Annual Mid-Winter Radiological Conference, sponsored by the Los Angeles Radiological Society, will be held at the Biltmore Hotel, Los Angeles, California, Saturday and Sunday, February 23 and 24, 1957.

An outstanding program of pertinent interest has been arranged, and the guest speakers will be Dr. John Caffey, New York; Dr. Johan Frimann-Dahl of Ulleval Hospital, Oslo, Norway; Dr. Merrill C. Sosman, Boston; and Professor Brian W. Windeyer, University of London and Middlesex Hospital, London, England.

The conference fee of $20.00 includes two luncheon meetings featuring questions and answers. A banquet ($7.50 per plate), preceded by cocktails will be held Saturday evening. Reservations may be made through Dr. Louis J. Bonann, 1245 Glendon Avenue, Los Angeles 24, California.

Courtesy cards will be available to residents in Radiology and radiologists in the Armed Forces by advance registration, with reduced tariff for the luncheons and banquet. Hotel reservations should be made promptly through the Convention Manager, Biltmore Hotel, Los Angeles, California.

SPECIAL FELLOWSHIPS AVAILABLE THROUGH AEC

The United States Atomic Energy Commission has made available a number of special fellowships for the academic year 1957–1958 in a relatively new field of scientific endeavor closely related to the atomic-energy program—radiological physics—according to an announcement by the Oak Ridge Institute of Nuclear Studies, which administers the fellowships for the Commission.

The radiological physics program provides for an academic year of formal courses at one of four universities to which fellows may be assigned, followed by a transfer to a corresponding cooperating AEC installation, where fellows train for approximately three months in applied health physics. The four centers of the program are at the University of Rochester, in cooperation with Brookhaven National Laboratory; the University of Kansas and the University of Washington, in cooperation with the Hanford Works; and Vanderbilt University, in cooperation with Oak Ridge National Laboratory. The programs at all four centers will include training in modern physics, radiation biology, radiation instrumentation, industrial hygiene and toxicology, biology, and research. A total of 75 fellowships is being offered.

Basic stipend for AEC fellows is $2500, with an additional $350 allowed for spouse and $350 for each dependent child. Fellowship awards include payment of normal tuition and fees required by the university, and a travel allowance for the fellow (not dependents) from the place of application to his assigned university. Radiological physics fellows receive an allowance for travel between the university and the cooperating laboratory.

Additional information and application blanks may be obtained from the Fellowship Office, University Relations Division, Oak Ridge Institute of Nuclear Studies, P.O. Box 117, Oak Ridge, Tennessee. Completed applications, supporting letters of reference, and transcripts must reach the Institute not later than February 15, 1957, to insure consideration. Appointments will be made on or about March 15, 1957.

RÖNTGEN'S X-RAY TUBE AT SMITHSONIAN INSTITUTION

One of the first x-ray tubes employed by Wilhelm Konrad Röntgen arrived in Washington, D.C. on August 24, 1956, where it was placed on permanent exhibition at the United States National Museum, Smithsonian Institution. The tube was purchased from a private owner in Germany and presented to the Smithsonian Institution by the General Electric Company's X-ray Department of Milwaukee, Wisconsin.

The newly acquired apparatus was Röntgen's third x-ray tube. His first two tubes are preserved, respectively, at the Physical Institute in Würzburg, Germany, and at the Deutsches Museum, Munich.

COURSE ON CLINICAL USE OF RADIOACTIVE ISOTOPES

A course on the clinical use of radioactive isotopes will be given by Dr. S. Feitelberg and Dr. Edith H. Quimby of the Department of Radiology, Columbia University, New York City. The dates of the course are June 3, 1957 through June 28, 1957, Monday through Friday, 9–12 A.M. and 1–5 P.M. The fee for the course is $250.00.

This is a full-time course which includes lectures, clinical rounds, and experimental laboratory exercises as well as clinical laboratory measurements on patients and on specimens. In addition to the listed instructors, sixteen invited lecturers from the New York area will present topics in their special fields.

Lectures. Physics of radioactive isotopes and interaction of radiation and matter. Techniques and measurements of radiation and of radioactive isotopes. Clinical use of radioactive iodine in thyroid disease. Therapeutic use of radioactive phosphorus and radioactive gold. Diagnostic use of miscellaneous isotopes (determination of blood

volume, localization of brain tumors, radioactive sodium and radioactive iron studies). Organization of radioisotope work in a hospital.

Laboratory Work. Thirteen afternoon sessions devoted to experiments on basic methods of radioisotope measurements, on techniques used in clinical diagnostic work and experience with equipment actually used in clinical procedures.

Enrollment in the course will be limited to twenty.

SPECIAL EXAMINATION FOR CERTIFICATION IN NUCLEAR MEDICINE

Within the very near future, a special examination for certification in Nuclear Medicine will be offered to diplomates in Radiology and Therapeutic Radiology. Those interested must apply to the American Board of Radiology before February 15, 1957.

B. R. Kirklin, M.D.
Secretary
Kahler Hotel Building
Rochester, Minnesota

DR. LAWRENCE REYNOLDS AWARDED GOLD MEDAL

Dr. Clarence E. Hufford, Toledo, Ohio, President of the Radiological Society of North America, at the recent annual meeting on December 11, 1956, presented to Dr. Lawrence Reynolds, Editor of this Journal, the Gold Medal of the Society. Fellow Editor of *Radiology*, Dr. Howard P. Doub, assisted in the bestowal of the coveted Medal, awarded annually by the Radiological Society of North America for outstanding contributions to the science of radiology.

CANADIAN ASSOCIATION OF RADIOLOGISTS TO MEET

The 20th Annual Meeting of the Canadian Association of Radiologists will take place from Monday, January 14 to Thursday, January 17, in the Sheraton Mount Royal Hotel, Montreal.

Major features of this anniversary meeting will be the five-part report on Cobalt^{60}, addresses of welcome by His Worship, Mayor Jean Drapeau, and Dr. Marc Trudel, president of the College of Physicians and Surgeons of the Province of Quebec, the Gordon Richards Memorial Lecture, and a civic reception to be held at the Chalet on beautiful St. Helen's Island.

The annual meeting of the Association will take place on Monday, January 14, at 7:30 P.M., at which time reports will be given and officers and directors elected. The annual dinner will be on Wednesday, January 16, at 7:30 P.M.

An impressive list of papers will be presented during the scientific sessions of this annual meeting.

Radiologists from the United States of America are cordially invited to attend.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

Intravenöse Cholangiographie; Grundlagen, Technik, Ergebnisse. Von Priv.-Doz. Dr. Th. Hornykiewytsch, Giessen. Cloth. Price, DM 54.—. Pp. 159, with 110 illustrations. Georg Thieme, Herdweg, 63, Stuttgart, Germany, 1956.

In this timely and excellent book the author draws upon a wealth of material derived from more than 3,000 patients studied by means of intravenous cholangiography. With noteworthy precision and thoroughness the author includes a comprehensive study of the normal and abnormal anatomy and histology, normal and abnormal physiology, pharmacology, and the roentgen appearance of the normal and abnormal biliary tract. The indications and contraindications as well as the pitfalls and sources of error are stressed. Laminagraphy is used practically routinely. The text is clear and well documented. Several tables and illustrations and a large number of reproductions of excellent roentgenograms accompany the text.

While there may be room for a variance of opinion in the interpretation of certain roentgen findings, especially in some phases of disturbed physiology, one is impressed by the worthwhileness of the work and the author's valuable contribution—the first book to be published to date on this subject.

The author very ably brings out the fallacy of the so-called postcholecystectomy syndrome, stressing the fact that this represents essentially a late recognition of disease existing prior to cholecystectomy. The evolution and development of hypertrophy and stenosis of the sphincter of Oddi and its clinical-roentgenologic significance are also emphasized.

The book is highly recommended to radiologists and gastroenterologists, as well as to surgeons and internists who are especially interested in the problems of the biliary and digestive tracts.

William H. Shehadi, M.D.

Hematology. By Cyrus C. Sturgis, M.D., Professor of Internal Medicine, Chairman of the Department of Internal Medicine, University of Michigan Medical School, and Director of the Thomas Henry Simpson Memorial Institute for Medical Research, University of Michigan, Ann Arbor, Michigan. Second edition. Cloth. Price, $19.50. Pp. 1,222, with 79 illustrations and 44 tables. Charles C Thomas, Publisher, 301 East Lawrence Avenue, Springfield, Illinois, 1955.

Significant developments in the field of hematology during the past seven years have been included in this completely revised second edition. The author calls attention to the difficulty of maintaining completeness in a textbook on hematology, or for that matter, on any division of medicine that is as fast moving. He emphasizes the importance of including the significant facts of each hematologic disorder that have been firmly established, as well as an evaluation of new advances. Wisely, the historical discussions have been retained and the bibliography has been most carefully selected.

In this revision Dr. Sturgis has brought up to date his very authoritative and practical text on hematology.

William S. Reveno, M.D.

Pelvo-Spondylitis Ossificans; Rheumatoid or Ankylosing Spondylitis. By Ragnar Romanus, M.D., and Sven Ydén, M.D., Karolinska Sjukhuset and King Gustaf V's Research Institute, Stockholm, Sweden. Cloth. Price, $8.50. Pp. 162, with 55 illustrations. Year Book Publishers, Inc., 200 E. Illinois Street, Chicago 11, Illinois, 1955.

This monograph is described by the authors as a roentgenologic and clinical guide to the early diagnosis of Marie-Strümpell type of arthritis or rheumatoid spondylitis. Their material consists of 250 cases of the entity which have been extensively and repeatedly studied.

One half of the volume correlates roentgenographic signs and corresponding clinical symptoms in all parts of the body involved by the disease. Histologic studies are considered. The course of the disease, its variability, terminology, etiology and pathology are presented in an organized manner. The bibliography is extensive.

The second half of the monograph consists of

multiple illustrative roentgenograms of clinical material. These are well reproduced and graphically described.

The volume can be read with both profit and pleasure by people in all fields of medicine who are interested in the early diagnosis of rheumatoid spondylitis. The authors make their thesis a plea for early recognition and accomplish it with clarity and logic.

Frank P. Walsh, M.D.

Röntgendiagnostik des Hüftgelenks. Von Prof. Dr. Med. R. Glauner, Chefarzt der Röntgen-und Radiumabteilung am Marienhospital Stuttgart; und Doz. Dr. Med. Habil. W. Marquardt, Leitender Arzt der Orthopädischen Klinik, "Paulinenhilfe," Stuttgart. Cloth. Price, $13.60. Pp. 168, with 195 illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1956.

The main emphasis of this monograph is placed on illustrations of the various abnormalities and diseases in and around the hip joint. The illustrations are excellent, and follow-up series of various conditions are quite documentative. The text is short, and so is the bibliography.

Richard Schatzki, M.D.

Bone and Joint X-Ray Diagnosis. By Max Ritvo, M.D., Assistant Clinical Professor of Radiology, Harvard Medical School; Instructor in Radiology, Tufts College Medical School; Lecturer on Radiology, Boston University School of Medicine; Director, Department of Radiology and Roentgenologist-in-Chief, Boston City Hospital. Cloth. Price, $20.00. Pp. 752, with 398 illustrations. Lea & Febiger, Washington Square, Philadelphia 6, Pennsylvania, 1955.

This textbook covers the extremely wide field of bone, joint and associated soft tissue roentgen diagnosis. It is a tribute to the author's wide knowledge of general radiology and applied medicine, for, although he draws freely from the medical literature and texts, much of his writing is colored by his long personal experience in the field. The chapters of the book cover the entire host of hereditary, congenital, and developmental skeletal disturbances; all traumatic lesions with reference to individual bones; infections; glandular, nutritional and metabolic disturbances; bone toxicology; tumors; joint and periarticular pathology; neuroradiology of the spinal canal; and a wide variety of loosely connected subjects listed under soft tissue roentgenography. Historical data, clinical aspects, roentgenologic manifestations and current management are discussed in most instances, although the amount of space allotted to each subject is necessarily limited. A brief introductory chapter scans the basic principles of roentgenography and covers the entire field of osteology. There is a liberal use of excellent, positive roentgenographic reproductions throughout the text. The arrangement of differential diagnostic points in tabular form is helpful for teaching purposes.

This book should prove a welcome and useful addition to the library of the young radiologist. Both orthopedic and radiologic residents will find it an excellent source for study and review of this vast subject. The well established radiologist will probably find a considerable duplication of material which is already present on his book shelves. This disadvantage is offset by the excellent presentation of a number of relatively little known and rare syndromes and conditions affecting bones and soft tissues which would save countless hours of perusing the literature when such cases arise.

Arch H. Hall, M.D.

Tumors of Bone; A Roentgenographic Atlas. By Bradley L. Coley, M.D., F.A.C.S., Attending Surgeon, Bone Service, Memorial Center for Cancer and Allied Diseases; Associate Professor of Clinical Surgery, Cornell University Medical College, New York; and Norman L. Higinbotham, M.D., C.M., F.A.C.S., Associate Attending Surgeon, Bone Service, Memorial Center for Cancer and Allied Diseases; Assistant Professor of Clinical Surgery, Cornell University Medical College, New York. Cloth. Price, $19.00. Pp. 216, with 172 illustrations. Paul B. Hoeber, Inc., 49 East 33rd Street, New York 16, New York, 1953.

This atlas is similar in format to the previous volumes in this series. It presents roentgenograms of various stages and types of malignant bone lesions, as well as a short section on benign bone tumors and non-neoplastic bone lesions simulating bone tumors. Unfortunately, very little of the clinical history or course is given, and in many cases there is no clinical information at all. A few of the cases have the gross pathologic specimen demonstrated, but little histology is included.

Each section of roentgenograms is prefaced by a brief summary of the pertinent roentgenographic features of the tumor. Again, as in the roentgenogram captions, no clinical history or course is given, nor are ages mentioned. Such information might be of value in the differential diagnosis. Differential diagnosis from other bone tumors is considered very briefly, but the last chapter contains a good discussion of the differential diagnosis of non-neoplastic conditions simulating bone tumors. No mention is made of the benign metaphyseal cortical defects somewhat similar to nonossifying fibroma.

Many of the reproductions in this atlas are of high quality. All are shown as negatives. The book is valuable as a roentgenographic atlas, since it shows the wide variety of roentgenographic features in any one particular tumor, as well as in multiple confusing non-neoplastic lesions.

M. MOTTRAM, M.D.

CANCER OF THE LUNG; PATHOLOGY, DIAGNOSIS, AND TREATMENT. By Milton B. Rosenblatt, M.D., Associate Professor of Medicine, New York Medical College, and Visiting Physician and Chief of Chest Clinic, New York City Hospital; and James R. Lisa, M.D., Director, Pathology Service, New York City Departments of Hospitals, Director of Laboratories and Pathologist, Doctors Hospital. Cloth. Price, $15.00. Pp. 330, with numerous illustrations. Oxford University Press, 114 Fifth Avenue, New York 11, New York, 1956.

In 300 pages the authors, with the aid of other contributors, summarize the problem of cancer of the lung. Though much of the material compiled in the book is already familiar to the radiologist and others who deal with neoplasms of the lung, the authors add some of their own material.

The usual statistics, the depressing results, and the standard syndromes are all reviewed. The roentgenograms illustrate typical manifestations. Surgeons have contributed sections on the techniques of performing lobectomy and pneumonectomy.

The book includes an excellent short chapter on exfoliative cytology by Dr. George N. Papanicolaou and Dr. N. Chandler Foot. A summary of the radiation techniques available for palliation, written by Dr. Bernard Roswit, is also noteworthy. Each chapter is followed by a rather extensive bibliography.

HENRY J. KLOS, M.D.

CANCER; A MANUAL FOR PRACTITIONERS. Third edition. Cloth. Price, $2.00. Pp. 321. Published by American Cancer Society (Massachusetts Division), Inc., 462 Boylston Street, Boston 16, Massachusetts, 1956.

The latest revision of this thoroughgoing review consists of 50 chapters by almost as many authorities, each dealing with some phase of the cancer problem. The discussions are terse, objective and factual, and are pointed toward early recognition of cancer.

This manual should serve as a useful, informative guide for the practitioner in the management of his cancer patients.

WILLIAM S. REVENO, M.D.

DIE RÖNTGENBILDANALYSE. EINE RÖNTGENDIAGNOSTISCHE ANLEITUNG FÜR STUDIERENDE UND ÄRTZE. By Prof. Dr. Erich Saupe, and Prof. Dr. W. Teschendorf. Third edition. Cloth. Price, DM 49.50. Pp. 283, with numerous illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1956.

Since Saupe published this book in 1942, it has occupied an important place in the libraries of many medical students, residents in radiology, and practicing physicians. It was planned to present the basic principles for the interpretation of roentgenograms and to describe methods of reporting diagnoses. These ideas define the entire book and therefore it should not be considered as a textbook of radiology.

After Saupe's death Teschendorf revised this valuable work. He kept Saupe's basic outline and tried to complete it by including recent advances in the field of radiology, as Saupe might have done it himself. Especially interesting paragraphs are: Theory of Projection, Summation Effects, Enlargement Technique, Mistakes in Developing and Material Defects, Functional Diagnosis, Analysis of Bone Fractures, Tumors, Anomalies, Roentgen Diagnosis of the Chest, Genitourinary Tract, Gallbladder, Liver and Spleen, etc. Special methods described include: Kymography, Elektrokymography, Angiocardiography, Laminagraphy, Bronchography, Stereoscopy and Roentgencinematography. The final chapters discuss: How to Study Radiology, Position of Radiology in Medicine, and How To Organize an X-Ray Department.

Teschendorf also considers the clinical theory of diseases and their differential diagnosis. The discerning reader will find here, in concentrated

form, an excellent manual of modern radiology which will help him to avoid mistaken interpretations, will lead him into the extensive field of radiology, and will show him the present diagnostic possibilities.

The book contains many good illustrations and a valuable bibliography of the borderlands and the handbooks of radiology. It can, without reservation, be recommended for general use.

MARTIN DONNER, M.D.

MEDICAL PHOTOGRAPHY; RADIOGRAPHIC AND CLINICAL. By T. A. Longmore, Hon. F.S.R., Principal, Kodak School of Medical Radiography and Clinical Photography; Hon. Editor of "Radiography," Member of Council, Education Committee and Board of Examiners of the Society of Radiographers; Late Senior Instructor in Radiographic and Medical Photography at the Royal Army Medical College, etc. Fifth edition. Cloth. Price, $15.00. Pp. 1,000, with 319 figures and 76 plates. Focal Press Ltd., 31 Fitzroy Square London W. 1, England, 1955.

"Medical Radiography," now in its fifth edition, has become an important text to the medical roentgenographer and photographer. Although its circulation has largely occurred in Great Britain, many in the United States who employ such texts have become cognizant of its value. The chief source material is related to the photographic aspects of roentgenography and medical photography. The chapters covering data relative to kymography, stereoroentgenography and electrography have been revised and expanded and consitute excellent reference material. The sections on medical still and motion photography contain an abundance of up-to-date material of interest to all photographers. An entirely new section pertaining to color photography has been added to the basic photographic text which has been revised and enlarged to embrace recent advances, particularly in flash techniques.

ARTHUR W. FUCHS

BOOKS RECEIVED

PROGRESS IN NUCLEAR ENERGY. SERIES VII. MEDICAL SCIENCES. Volume I. Edited by J. C. Bugher, The Rockefeller Foundation, New York; J. Coursaget, C. E. A., Saclay; and J. F. Loutit, Medical Research Council, A. E. R. E., Harwell. Cloth. Price, $6.00. Pp. 165. McGraw-Hill Book Company, Inc., 330 West 42nd Street, New York 36, New York, 1956.

IIe CONGRÈS INTERNATIONAL DE RADIOPHOTOGRAPHIE MÉDICALE. PARIS—4–7 AVRIL, 1956. RAPPORTS. Paper. Price, 2.500 fr. Pp. 356, with figures and tables. Masson et Cie, Éditeurs, 120, boulevard Saint-Germain, Paris-6e, 1956.

CLINICAL ROENTGENOLOGY. VOLUME IV. THE DIGESTIVE TRACT, THE GALL BLADDER, LIVER AND PANCREAS, THE EXCRETORY TRACT AND SPECIAL STUDIES EMPHASIZING DIFFERENTIAL CONSIDERATIONS. By Alfred A. de Lorimier, M.D., Radiologist, Saint Francis Memorial Hospital, San Francisco, California; Henry G. Moehring, M.D., Radiologist, Duluth Clinic, Duluth, Minnesota; and John R. Hannan, M.D., Radiologist, Cleveland, Ohio. Cloth. Price, $24.50. Pp. 764, with 1,112 illustrations. Charles C Thomas, Publisher, 301 East Lawrence Avenue, Springfield, Illinois, 1956.

DISEASES OF THE HEART. By Charles K. Friedberg, M.D., Attending Physician, The Mount Sinai Hospital, New York; Associate Clinical Professor of Medicine, College of Physicians and Surgeons, Columbia University. Second edition. Cloth. Price, $18.00. Pp. 1,161, with 157 illustrations. W. B. Saunders Company, 218 West Washington Square, Philadelphia 5, Pennsylvania, 1956.

THERAPEUTIC USE OF ARTIFICIAL RADIOISOTOPES. Edited by Paul F. Hahn, Ph.D., Cancer Research Laboratories, Nashville, Tennessee. Cloth. Price, $10.00. Pp. 414, with numerous illustrations. John Wiley & Sons, Inc., 440 Fourth Avenue, New York 16, New York, 1956.

RÖENTGENKUNDE UND KLINIK VERTEBRAGENER KRANKHEITEN. Edited by Prof. Dr. med. Herbert Junghanns. Cloth. Price, DM 24.50. Pp. 132, with 62 illustrations. Hippokrates-Verlag, Stuttgart, Germany, 1956.

THE XIV JAPAN MEDICAL CONGRESS. APRIL 1–5, 1955, KYOTO. PART I AND PART II. Edited by The Bureau of Archives, The XIV Japan Medical Congress at the Faculty of Medicine, Kyoto University. Paper. Price, $6.00. Pp. 733, with numerous illustrations. The Bureau of Archives, The XIV Japan Medical Congress, Department of Pathology, Faculty of Medicine, Kyoto University Yoshida, Sakyo-ku, Kyoto, Japan, 1956.

PROGRESS IN RADIOBIOLOGY. PROCEEDINGS OF THE FOURTH INTERNATIONAL CONFERENCE ON RADIOBIOLOGY HELD IN CAMBRIDGE ON 14TH TO 17TH AUGUST, 1955. Edited by Joseph S. Mitchell, Barbara E. Holmes, and Cyril L. Smith, Department of Radiotherapeutics, University of Cambridge, Cloth. Price, $12.75. Pp. 557, with numerous illustrations. Charles C Thomas, Publisher, 301 East Lawrence Avenue, Springfield, Illinois, 1956.

CHEMOSURGERY IN CANCER, GANGRENE AND INFECTIONS. By Frederic E. Mohs, B.Sc., M.D.,

Associate Professor of Chemosurgery, University of Wisconsin Medical School; Head of the Chemosurgery Clinic, State of Wisconsin General Hospital; Research Associate, McArdle Memorial Laboratory for Cancer Research, Madison, Wisconsin. Cloth. Price, $13.50. Pp. 305, with 225 illustrations. Charles C Thomas, Publisher, 301 East Lawrence Avenue, Springfield, Illinois, 1956.

Tenth Annual Report of the Oak Ridge Institute of Nuclear Studies. Operating Under Contract with the United States Atomic Energy Commission and the National Science Foundation, June 30, 1956. Paper. Pp. 113, with numerous illustrations. Oak Ridge Institute of Nuclear Studies, P.O. Box 117, Oak Ridge, Tennessee, 1956.

Berättelse from Styrelsen för Cancerföreningen I Stockholm över Verksamhetsåret 1955. Redogörelser för Verksamheten å Radiumhemmet, Radiopatologiska Institutionen Och Radiofysiska Institutionen Vid Konung Gustaf V:S Jubileumsklinik I Stockholm under år 1955; Index of Paper Published at the Radiumhemmet, the Institute of Radiopathology and the Institute of Radiophysics; Report of Dec. 31st, 1955, on Patients Registered at the Radiumhemmet 1921–1955. Paper. Pp. 145, with tables. K. L. Beckmans Boktryckeri, Stockholm, 1956.

Die Lungentuberkulose; Diagnose und Therapie. Von Prof. Dr. Med. P.-G. Schmidt, Waldbreitbach. Third edition. Cloth. Price, DM 58.—. Pp. 384, with 234 illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1956.

Pelvimetry. By Herbert Thoms, M.D., Emeritus Professor of Obstetrics and Gynecology, Yale University School of Medicine, New Haven, Connecticut. Cloth. Price, $5.00. Pp. 120. with 45 illustrations. Paul B. Hoeber, Inc., 49 East 33rd Street, New York 16, New York, 1956.

Clinical Pathology; Application and Interpretation. By Benjamin B. Wells, M.D., Ph.D., Director of Clinical Investigation, The Lynn Clinic, Detroit. Former Professor of Medicine and Chairman of the Department of Medicine, Creighton University School of Medicine, Omaha. Second edition. Cloth. Price, $8.50. Pp. 488, with 25 illustrations. W. B. Saunders Company, 218 West Washington Square, Philadelphia 5, Pennsylvania, 1956.

La Radiologie Clinique de l'Oesophage. Par Marcel Brombart, Chef du Service de Radiodiagnostic à la Clinique César de Paepe, Bruxelles. Cloth. Price, 5.000 fr. Pp. 466, with 337 illustrations. Masson et c^ie, Éditeurs, 120 boulevard Saint-Germain, Paris 6^e, France, 1956.

Diagnostic Différentiel Radiologique des Ulcérations Gastriques. Par Georges Candardjis. Paper. Price, 1.500 fr. Pp. 188, with 92 illustrations. Masson et C^ie, Éditeurs, 120 boulevard Saint-Germain, Paris 6^e, France, 1956.

Études sur la Précision en Radiodiagnostic. Techniques d'un Nouveau Craniographe. Notation Angulaire de l'Incidence. Par G. L. Dulac, Ancien chef de service à la Fondation Curie. Paper. Price, 1.300 fr. Pp. 134, with 47 illustrations. Masson et C^ie, Éditeurs, 120 boulevard Saint-Germain, Paris 6^e, France, 1956.

Technique du Radiodiagnostic. Par J. Garcia-Caldéron et G. Ledoux-Lebard. Second edition. Cloth. Price, 7.800 fr. Pp. 1,418, with 746 illustrations. Masson et C^ie, Éditeurs, 120 boulevard Saint-Germain, Paris 6^e, France, 1956.

Le Traitement des Angiomes Chez les Enfants. Par Simone Laborde, Chef de Service Honoraire de l'Institut G. Roussy. Paper. Price, 1.850 fr. Pp. 174, with 71 illustrations. Masson et C^ie, Éditeurs, 120 boulevard Saint-Germain, Paris 6^e, France, 1956.

Grundlagen und Praxis der Bewegungsbestrahlung. Band II. Von H. Langendorff, W. J. Lelbach, R. Janker, K. Rossmann. Cloth. Pp. 212, with numerous illustrations. Verlag W. Girardet, Neumarkstr. 50, Wuppertal-Elberfeld, Germany, 1955.

Action des Radiations Ionisantes sur l'organisme. Par A. Lacassagne, Professeur honoraire au Collège de France, Directeur honoraire de l'Institut du Radium (Section biologique et thérapeutique); et G. Gricouroff, Chef du Service d'Histopathologie de la Fondation Curie (Institut du Radium de l'Université de Paris). Second edition. Paper. Price, 1.500 fr. Pp. 198, with 17 illustrations. Masson et C^ie, Éditeurs, 120 boulevard Saint-Germain, Paris 6^e, France, 1956.

Strahlendosis und Strahlenwirkung. Tafeln und Erläuterungen—Unterlagen für den Strahlenschutz. Von Prof. Dr. B. Rajewsky, Frankfurt/M. Second edition. Plastic. Price, $8.60. Pp. 486, with tables and charts. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1956.

Medizinische Röntgentechnik. I. Medizinischer Teil. Skelettaufnahmen und Organuntersuchungen. Von Prof. Dr. Herbert Schoen, Karlsruhe, Second edition. Cloth. Price, DM 29.70, Pp. 347, with 543 illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1956.

Principles of Chest X-Ray Diagnosis. By George Simon, M.D., F.F.R., Assistant Director, X-ray Department, Brompton Hospital, London; Radiologist, St. Bartholomew's Hospital, London; Demonstrator, Radiological Anatomy, St. Bartholomew's Hospital, London, Cloth. Price, 50s. Pp. 183, with 162 illustrations. F. A. Davis Company, 1914 Cherry Street, Philadelphia 3, Pennsylvania, 1956.

Vol. 77, No. 1

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Head

Salvini, L., and Collo, G. L'emicraniosi di Brissaud-Lereboullet. (Hemicraniosis of Brissaud-Lereboullet.) *Ann. radiol. diag.*, 1956, *29*, 165–178. (From: Istituti di Radiologia e del Radio dell'Università, Bologna, and Istituti di Radiologia e del Radio degli Spedali Civili, Brescia, Italy.)

This is an extremely rare condition which was first described in 1903. It is characterized by an asymmetry of the skull due to enlargement of the frontal or sphenoid bone on one side which manifests itself either at birth or in early infancy. The prominence of the frontal bone becomes more progressive until intracranial symptoms develop and finally prove fatal in early life. The thickening of the bone involved affects both the outer and inner tables. The underlying dura contains tumoral masses resembling meningiomas.

The authors present 2 cases which fall in this category and in one of the cases biopsy material was obtained. Roentgenographic reproductions included in this article show the extensive localized hyperostosis involving both tables of the skull.

The authors feel that the bone involvement is an embryologic process which later is followed by a sarcomatous degeneration of the dura.—*Peter E. Russo, M.D.*

Verbiest, H. Contribution to the methods of filling the posterior fossa and the adjoining cervical subarachnoid space with small quantities of air. *Brit. J. Radiol.*, Aug., 1956, *29*, 440–444. (From: Neurosurgical Department, University Clinic, Utrecht, Holland.)

The author describes a method of visualizing the posterior fossa and cervical subarachnoid space in cases where an erect position is difficult to maintain or when it causes a fall in blood pressure or even syncope. The patient is placed in the prone position on a tilting table with the neck flexed, if necessary by placing a pillow under the chest and shoulders. Thirty cc. of air is then introduced by cisternal puncture. Changes in posture are produced by increasing the inclination of the table. When the table is tilted to 30 degrees this air fills the dorsal part of the cervical subarachnoid space. With an inclination of 45 degrees it passes over the posterior and superior surface of the cerebellum. Additional inclination to about 60 degrees may result in filling of the anterior part of the cisterna magna and the fourth ventricle.

During ventriculography when air has been introduced into a lateral ventricle, several maneuvers have been described to move it into the fourth ventricle. If there is absence or incompleteness of obstruction, the air may escape entirely into the spinal subarachnoid space. When this happens, if the patient is placed in the horizontal supine position with the neck flexed and the table is then inclined to 50–60 degrees, the fourth ventricle is frequently visualized. It is also a good method to demonstrate herniation of the cerebellar tonsils.—*Arthur E. Childe, M.D.*

Neck and Chest

Lubert, Mortimer, and Krause, George R. Total unilateral pulmonary collapse; a study of the roentgen appearance in the lateral view. *Radiology*, Aug., 1956, *67*, 175–185. (Address: M. Lubert, Mount Sinai Hospital, 1800 E. 105th St., Cleveland 6, Ohio.)

When a main bronchus to a lung becomes occluded, the final appearance in the lateral roentgenogram is independent of the side involved or the site of the endobronchial block. When other processes, not primarily or solely endobronchial, cause a unilateral pulmonary collapse the lateral view has essentially the same appearance as that resulting from a blocked bronchus. Even when the basic abnormality is obstructive emphysema of the opposite lung, the appearance in the lateral view is still the same.

The similarity in appearance is due to the action of similar forces on the same anatomic structures. The posterior portion of the mediastinum is fixed and rigidly anchored. There is slightly more mobility of the middle mediastinum. The tissues of the anterior superior mediastinum are relatively thin and offer little resistance to displacement. Any difference in pressure between the two halves of the thorax therefore exerts its maximum effect anteriorly. The overdistended lung "herniates through" anteriorly, and the heart and trachea shift to the side of lesser pressure. Thus the collapsed lung moves laterally, posteriorly and inferiorly. This sequence of events is responsible for the roentgen picture in total collapse of a lung. There is increase in size and radiolucency of the anterior mediastinum with posterior displacement of the heart and great vessels. There is a generalized increase in density postero-inferiorly. The margin of the collapsed lung is visualized if it is in contact with the opposite overexpanded lung at the retrocardiac space. In the presence of massive pleural effusion the mediastinum is displaced to the opposite side and the radiolucent shadow of the anterior mediastinum is obliterated.

Numerous reproductions of roentgenograms showing collapse, and several diagrams explaining the mechanism of unilateral collapse, are included.—*A. W. Sommer, M.D.*

Davis, Edgar W., Katz, Sol, and Peabody, J. Winthrop, Jr. Broncholithiasis, a neglected cause of bronchoesophageal fistula.

J.A.M.A., Feb. 18, 1956, *160*, 555–557. (Address: E. W. Davis, 1150 Connecticut Ave., Washington, D. C.)

A case of acquired nonmalignant esophagobronchial fistula is reported. A preliminary roentgenogram of the chest revealed a large mass of calcified lymph nodes in the right hilus. The history showed that the patient had coughed up "stones" and also liquids and ingested foods. Barium studies of the esophagus demonstrated barium in both the esophagus and trachea. Introduction of iodochlorol by means of a catheter in the esophagus, disclosed a fistula in the mid-esophagus. Surgical exploration confirmed the presence of a fistula 1 cm. in diameter and in length between the anterior surface of the esophagus and the right main bronchus, which was repaired.

Bronchoesophageal fistula occurs twice as often on the right side as on the left, coinciding with the more common location of a calcified lymph node complex on the right side. In cases of bronchoesophageal fistula, investigation should be directed to roentgenographic or historic evidence of broncholithiasis before attributing the debilitated state to inoperable carcinoma. Introduction of iodized oil into the esophagus usually demonstrates the presence of fistulae, even if very small, and verification can usually be established by means of bronchoscopy.—*C. N. Borman, M.D.*

SAYMAN, M. ISMET. A new sign in the diagnosis of cardiac aneurysm and myocardial infarction; (respiratory phenomenon; effort test; paradoxical expansion of the heart). *Radiology*, Aug., 1956, *67*, 242–243. (Address: Denizcilik bankasi hastanesi, Röntgen mütehassisi, Istanbul-Galata, Turkey.)

A method of diagnosis of cardiac aneurysms and myocardial infarction based on roentgenologic examination of the heart in forced inspiration and expiration is presented. During forced inspiration in cardiac aneurysm the aneurysmal sac becomes distended and more prominent while in forced expiration the aneurysmal sac collapses, disappearing completely.

In myocardial infarction the area of infarction appears flattened or even depressed in forced inspiration while in forced expiration the line of infarction disappears completely. Following exertion, the pulsations above and below the area of infarction will be found to be increased in amplitude, while those in the area of infarction will have become reduced or even have disappeared altogether.

In total cardiac aneurysm there is an outward expansion of the left ventricular border and an increase in heart size in inspiration and an inward retraction of the left ventricular border with a reduction of heart size in expiration.—*Harold C. Hamilton, M.D.*

SCHORR, S., DREYFUSS, F., and FRÄNKEL, M. Evaluation of the recumbent esophagogram in the early detection of left atrial enlargement. *Radiology*, Aug., 1956, *67*, 186–194. (Address: S. Schorr, Diagnostic X-Ray Department, Hadassah University Hospital, Jerusalem, Israel.)

This study evaluating the recumbent esophagogram as an early sign of left atrial enlargement is based on 311 cases, including 173 cases with definite clinical evidence of mitral valvular disease, 17 with probable mitral valvular disease, 7 with various types of congenital heart disease, and a control group of 114 patients without clinical evidence of heart disease of any kind. Fluoroscopy of the chest was performed, and posteroanterior teleroentgenograms and left and right anterior oblique films were obtained. Erect and recumbent right lateral esophagograms were obtained under fluoroscopic control. The right lateral view was preferred to the right anterior oblique view, since comparison of films is difficult when the angle of obliquity varies even slightly.

In the group of 114 patients with clinically normal hearts both the erect and recumbent esophagograms were normal, except in 8 per cent, where a positive esophagogram, erect or recumbent, was obtained. Sixty-two of the 173 patients with definite clinical evidence of mitral valvular disease had positive esophagograms, both erect and recumbent, while in 27 of this group both the erect and recumbent esophagogram were negative. In 84 cases with mitral valvular disease diagnosed clinically, only the recumbent esophagogram was positive, and in 36 of these cases there were no other corroborative roentgen signs of the condition. Nine of the group of 17 patients with probable mitral valvular disease had negative esophagograms and 8 had positive esophagograms. No patient in this study with a positive erect esophagogram had a negative recumbent esophagogram. This study confirms the previous opinion of the authors that the recumbent esophagogram is a reliable and sensitive indicator of slight left atrial enlargement.—*Harold C. Hamilton, M.D.*

SANTAGADA, A., and PIAZZI, M. Contributo allo studio radiologico del gozzo intratoracico mediante stratigrafia assiale transversa. (Radiologic studies of intrathoracic goiters by means of transverse axial stratigraphy.) *Ann. radiol. diag.*, 1956, *29*, 186–213. (From: Istituti di Radiologia e del Radio dell'Università, Bologna, Italy.)

The authors have used transverse axial laminagraphy to study mediastinal or intrathoracic goiters to better determine their exact size and position. The air filled trachea and the barium opacified esophagus serve as excellent landmarks and the

displacement or distortion can be well recorded in studies of these organs.

Several such cases with very good illustrations are included in this article. The authors feel that the addition of pneumomediastinum combined with this procedure gives no additional information and therefore is not necessary.

Although this work on the subject is excellent it may become useful only in the more difficult diagnostic cases. More practical methods will give sufficient information, I believe, in the average case.—*Peter E. Russo, M.D.*

Gillis, D. A., Reynolds, D. P., and Merritt, J. W. Chemodectoma of an aortic body. *Brit. J. Surg.*, May, 1956, *43*, 585–587. (From: The Institute of Pathology of Dalhousie University and the Children's Hospital, Halifax, Nova Scotia.)

Nonchromaffin paraganglioma or chemodectoma may arise in many different areas including the middle ear, in association with the jugular bulb, the upper cervical region and a few other areas. The authors report one which occurred in a seven-year-old boy arising from the aortic body. It was removed surgically and subsequently recurred on several occasions and was quite radioresistant. There was never any evidence of distant metastases. The roentgenographic appearance was that of a sharply circumscribed density in the right lower perimediastinal region. The original tumor measured 4 cm. in diameter.

The differential diagnosis should include any middle or anterior mediastinal tumor.—*George L. Sackett, Jr. M.D.*

Abdomen

Chérigié, E. (Paris, France.) La radiologie du grêle inflammatoire (signes fonctionnels et organiques des affections inflammatoires). (The radiology of small bowel inflammations; functional and organic signs of inflammatory affections.) *J. de radiol. et d'électrol. & Arch. d'électric. méd.*, March–April, 1956, *37*, 133–144.

Lack of specific roentgenographic signs, the uncertainty and vast overlapping of signs and symptoms concerning the functional and organic disorders, and the difficulty in duplicating roentgen images (because of motility and superimposition of loops) render the study of small intestinal pathology quite complex and moot. Thus, after having examined more than 20,000 small intestine roentgenograms, the author often feels hesitant as to the interpretation of certain images.

Under the functional signs are listed motility, tonicity, segmentation, residual images (scattering effect), retention of gas and of liquids. It is reasonable to expect hypomotility of small bowel segments in the first days of an inflammatory affection because of mucosal edema. However, this being very transient, most authors find the succeeding phase, *i.e.*, hypermotility, as one of the earliest signs. Thus, barium passes rapidly through the small bowel, especially through the jejunum, reaching the cecum within one half hour. This sign is of value if constant and if other factors such as chilling, emotional upset and hyperthyroidism can be ruled out. It is of particular importance if mucosal pattern alterations can also be demonstrated concomitantly. If the inflammatory process continues on, or is aggravated, then an irregular motility and marked retardation of the passage of barium through the small bowel can be noted. Hypertonicity and an apparent transient reduction of the caliber of the small bowel loops are demonstrable with hypermotility. These signs are more clearly noted along the ileal loops. When deeper layers of small bowel are involved, hypotonicity can be noted.

Abnormal segmentation (zones of spasm alternating with zones of unusually enlarged loops) indicates serious affection of the small intestine. The scattering effect, especially in jejunal segments, retention of gas and liquid are all signs indicating inflammatory changes of the mucosal lining. These functional signs are usually transient and are noted mostly in cases of deficiency disease and ordinary transient diarrhea. Exceptions are sprue and celiac disease, which are of particular gravity.

Early organic signs demonstrable by roentgen study are mucosal edema (blurring of contour of loops and of mucosal folds), mesenteric lymph adenopathy and hypertrophy of intestinal lymphatic follicles and of Peyer's patches (which are hard to demonstrate roentgenographically except for pseudopolypoid negative images at terminal loops of ileum along its free borders, and in cases of ulceration flat bottomed niches can be noted).

Late organic changes are those of fibrosclerosis, atrophy of muscles, with stenosis and at times complete obstruction of lumen of intestinal loops. These changes are irreversible.

The author found follicular ileitis, mostly at terminal ileum, concomitant with infectious disease such as severe measles, scarlet fever, pneumonia, Vincent's angina or parasitic infestation. Many of these cases presented symptoms of pseudoappendicitis due to lymphatic follicular hypertrophy. This hypertrophy can also be noted in early cases of intestinal tuberculosis and Crohn's disease (regional ileitis) and typhoid fever. More advanced stages of these diseases show stenotic and ulcerative changes. Perforations are quite prevalent in typhoid ulcerations.—*Jirair N. Sarian, M.D.*

Ramsay, Gordon S. The negative x-ray report in cancer of the colon. *Brit. J. Surg.*, May, 1956, *43*, 576–579. (From: Westminster

Hospital Teaching Group [The Gordon], London, England.)

The author studied 151 cases of carcinoma of the colon which were seen from 1947 to 1953. Seventy-five of these had had barium enema examinations and in 10 a negative report had been given. In 7 cases there was a delay of from two months to three years in making the correct diagnosis. In retrospect, there were minor abnormalities on several of the studies but the remainder were normal upon being reviewed. Almost all of the patients had a history suggestive of carcinoma of the colon, and in 2 a mass was felt.

The author urges, therefore, that in spite of a negative roentgenographic report an exploratory laparotomy be done if the history and clinical findings are suggestive of carcinoma.—*George L. Sackett, Jr., M.D.*

MELAMED, JACK L., and PARKER, MORRIS L. Cholecystogastric fistula; report of a case. *J.A.M.A.*, Feb. 11, 1956, *160*, 463–464. (Address: J. L. Melamed, 2950 W. Arthur Ave., Chicago, Ill.)

A case is reported of cholecystogastric fistula, one of the infrequent forms of internal biliary fistulas. A lamellated gallstone was revealed in the right upper quadrant on the first roentgen examination. Six months later a large lamellated defect was detected in the distal portion of the stomach by means of a barium meal study. A fistulous tract between the stomach and gallbladder was identified and air was demonstrated in the biliary system. At surgery, a stone measuring 3 by 4.5 cm. was verified. The distal end of the stone was projecting into the pylorus, with the main portion in the antrum of the stomach. —*C. N. Borman, M.D.*

LICHT, ERIK DE FINE. Arteriomesenteric obstruction of the duodenum in adult life and adolescence. *Acta radiol.*, June, 1956, *45*, 441–450. (From: Department of Radiology, Københavns Amtssygehus, Gentofte, Denmark.)

There are 2 groups of patients with arteriomesenteric obstruction. The first group consists of patients with congenital disorders, whereas in the second group the condition is acquired. Congenital defects vary in severity and include malrotation, errors in fixation, and "intrinsic" obstruction due to a defect in vacuolization of the lumen of the small intestine, resulting in an intestinal septum. Such an "intrinsic" defect, or partial atresia, gives rise to severe symptoms in early childhood. In older children and adults, the milder forms of errors in rotation and fixation may be responsible for symptoms.

Acquired duodenal stenosis may result from duodenal ulcer, periduodenitis, pericholecystitis, perforating biliary calculi, carcinoma of the ampulla of Vater or the pancreas, retroperitoneal tumors, and other lesions.

The author primarily discusses the milder forms of arteriomesenteric obstruction, the more severe forms causing high small intestinal obstruction and being more obvious. The roentgen changes seen in these milder forms consist in dilatation of the duodenum up to the duodenojejunal flexure, reversed peristalsis in the dilated portion, and abnormally violent kneading of the intestinal content in the dilated intestine. There may be stasis and even gastric dilatation.

The symptoms are manifold and include fullness, belching, discomfort or depression after meals, a sensation of abdominal heaviness, and abdominal rumbling, often accompanied by pain, particularly after meals. "Nervous" anorexia was seen in 3 of the female patients.

Duodenojejunostomy is resorted to only when conservative measures, including bedrest and small meals, fail to control the milder cases.—*Michael J. Healy, M.D.*

ROSENBLUM, DAVID, and SCHWARTZ, SOLOMON. Intravenous cholangiography in the presence of jaundice. *Radiology*, Aug., 1956, *67*, 247–249. (Address: D. Rosenblum, Jewish Hospital of Brooklyn, 555 Prospect Place, Brooklyn 38, N. Y.)

Cholografin studies have been done in 26 jaundiced patients with successful common duct visualization in 5 (19 per cent). Both serum bilirubin determinations and the icterus index were obtained to establish the degree of jaundice. The former, however, appeared to give a more reliable criterion in determining the degree of jaundice.

In general there seemed to be 2 prerequisites to successful visualization of the common duct: (a) a waning jaundice, and (b) icterus index and serum bilirubin values within certain limits. Values not exceeding 12 for the icterus index and 1.7 for serum bilirubin, especially in a waning jaundice, offered the best chance of successful duct demonstration.

The use of a spasmodic such as morphine or demerol is mentioned as a possible way of increasing the percentage of successful visualizations.—*Donald N. Dysart, M.D.*

THEANDER, GEORG. Precipitation of contrast medium in the gallbladder. *Acta radiol.*, Dec., 1955, *44*, 467–470. (From: Roentgen Department, Malmö, Allmänna Sjukhus, University of Lund, Lund, Sweden.)

A case is reported in which after two doses of 3 gm. telepaque, given on succeeding days, preliminary films, as well as films after the first dose, failed to show contrast medium concentration, but after the second dose opaque shadows were visible in the right upper quadrant. A week later, cholographic studies

with biligrafin showed that the opacities were within the gallbladder. Re-examination one month later failed to reveal opacities in the right upper quadrant, and no contrast medium concentration was present in the gallbladder.

The author believes the opacities in the gallbladder were a precipitate of telepaque. The risk of precipitation and stone formation should be considered in cholography, although no such case has been previously described in the literature.—*C. N. Borman, M.D.*

Stuart, Charles. Acute pancreatitis; preliminary investigation of a new radiodiagnostic sign. *J. Fac. Radiologists*, July, 1956, *8*, 50–58. (From: Fremantle Hospital, Perth, Western Australia.)

Cases of acute pancreatitis which as "acute abdomens" come to exploratory operation rarely require further immediate surgery than the laparotomy which confirms the diagnosis. If one can make the diagnosis preoperatively, initial conservative and nonsurgical management offers the greatest chance of survival. Heretofore diagnostic criteria have been: (1) macroscopic changes seen in the pancreas at operation or at autopsy; (2) a serum-amylase estimation of 200 or more Somogyi units in cases which present with abdominal pain; (3) a urinary diastase estimation of 50 or more Wohlgemuth units in cases with abdominal pain; and (4) to these, Stuart adds, the colon "cut-off" sign.

Anatomy: The transverse colon is connected to the posterior abdominal wall by the transverse mesocolon, a broad fold of peritoneum. After surrounding the transverse colon the layers of peritoneum join behind it and then continue to the anterior border of the pancreas, where they separate. The anterior surface of the pancreatic head is in contact with the transverse colon, its lower portion being covered with peritoneum continuous with the inferior layer of the transverse mesocolon. On its right border the head of the pancreas is lodged within the curve of the duodenum. Accordingly, an acute inflammatory process in the head of the pancreas would be expected to produce changes in the structures in its immediate neighborhood, either by mechanical causes or by edema. In cases of acute pancreatitis in which laparotomy is performed, a cellulitis spreading from the edematous head of the pancreas between the layers of the transverse mesocolon to reach the mesocolic border of the colon just to the left of the hepatic flexure is found. This results in a localized obstruction of a segment of the transverse colon which appears collapsed and devoid of gas content.

Roentgenologically in the normal plain film of the abdomen, it is usual to demonstrate the whole outline of the colon by its gas and/or fecal content, and the shadow of the transverse colon can almost always be traced across the midline.

Stuart's routine for all patients admitted to the hospital with signs and symptoms suggesting acute abdominal lesion is plain roentgenography of chest and abdomen as soon as possible. Films include: (1) posteroanterior erect chest; (2) posteroanterior erect abdomen; (3) posteroanterior or anteroposterior recumbent abdomen; and (4) abdomen in lateral decubitus position. Where perforated peptic ulcer is suspected on clinical grounds but no free intraperitoneal air is seen on the films, a Seidlitz powder is given to the patient for gas insufflation of the stomach, and roentgen examination is then repeated.

Stuart reviewed the roentgenograms of 6 patients with acute pancreatitis, confirmed by operative or laboratory findings. In the plain films of the abdomen there was in each case a central segment of transverse colon which could not be identified by either gas or fecal content. Both the hepatic and splenic flexures of the colon showed a well-defined gas content, and these appeared to be sharply "cut-off" from the central transverse colon segment. Gas in the stomach, which may be lying transversely, could be confused with gas in the colon, and it may be difficult to differentiate isolated loops of gas-distended small intestine in the center of the abdomen from gas-filled colon.

In differential diagnosis, one must bear in mind: (1) perforation of a peptic ulcer (one of the most common acute abdominal lesions); (2) mechanical small intestinal or large intestinal obstruction; and (3) cardiac or pulmonary disease (evidence of which may or may not be seen on the routine posteroanterior erect chest film).—*Samuel G. Henderson, M.D.*

Larsen, Kaare A., and Pedersen, Arne. Roentgenologic findings in the stomach and duodenum in cancer of the pancreas. *Acta radiol.*, June, 1956, *45*, 459–469. (From: Department of Roentgenology, Aker Sykehus, and the Department of Pathology, Ullevaal Sykehus, Oslo, Norway.)

A retrospective study of the roentgen findings in 31 cases of cancer of the pancreas was made by the authors. Of these cases, 18 were felt to show signs which possibly indicated a malignant condition of the pancreas (only 8 of these had been reported and diagnosed or diagnosis rendered probable).

From their survey, comparatively small impressions and defects represented the most constant finding. The authors used the following criteria in studying their cases: (1) Small tumors of the head and body of the pancreas: small impressions in the duodenum; compression of the choledochus and dilatation of the biliary ducts may give postbulbar impression; (2) large tumors of the head and body of the pancreas: large impression and displacements of duodenum; constrictions, pad sign; (3) tumor infiltration beyond the pancreas (in wall and lumen of duodenum): rigid, irregular mucous membrane

relief, irregular defects; star shaped retractions due to shrinking; ulceration; (4) metastasis to the lymph nodes: impressions in duodenum, especially on medial aspect of duodenal loop. With lymph node metastasis in the hilus hepatis, bulbar impressions may be present; and (5) large, irregular, retroperitoneal infiltrations and infiltrations of the gastrohepatic ligament: major defects and displacements; increase of the distance between the spinal column and duodenal loop; irregular retractions in the stomach and duodenum by shrinking; contractions.

The authors admit a diagnosis based on such findings is not pathognomonic, but they feel that with better roentgen studies of the duodenal area and a higher index of suspicion, diagnosis will improve.—*George A. Miller, M.D.*

Gynecology and Obstetrics

Thierstein, Samuel T., and Grier, Maurice E. Clinical pelvimetry in teaching centers. *Am. J. Obst. & Gynec.*, June, 1956, *71*, 1258–1263. (From: Department of Obstetrics and Gynecology, Creighton School of Medicine, Lincoln, Neb., and St. Elizabeth Hospital, Omaha, Neb.)

The study is made to determine what emphasis is placed on examination of the obstetrical patient's pelvis as taught to medical students, interns and obstetrical residents in the leading obstetrical teaching centers in the United States today.

External pelvic measurements are done in 74 per cent of the teaching centers. Quite extensive manual pelvic measurements are done in 89 per cent of the teaching centers, while very little stress is placed on them in 11 per cent of the centers.

Roentgen measurements are done on all primiparas in 8 per cent of the teaching centers. In the remaining 92 per cent of the centers roentgen measurements are done in 75 down to 5 per cent of all primiparas. Roentgen measurements are taken in the last six weeks of pregnancy in 94.2 per cent of the teaching centers and in early pregnancy in 5.8 per cent of the teaching centers.

The trend is toward using illustrated sheets in pelvic examination as an aid to teaching. An illustrated pelvic examination record has been prepared to combine the essential features of records used by several teaching centers. The characteristic of each entry of the pelvic examination is graded in 1 of 3 categories. Category 1 is a normal pelvis. Category 2 is a pelvis with moderate limitation in size. The pelvis of Category 3 has undesirable features in size and type.

The results of 12 entries are coded, the sum of the entries in each category being listed. As the shift in the entries is toward the right, the incidence of pelvic contraction and dystocia increases. The authors recommend that illustrated pelvic examination sheets be used and that external measurements be continued.—*Eugene J. McDonald, M.D.*

Muller, W. H. Otto pelvis; a report of two cases associated with labour. *J. Obst. & Gynaec. Brit. Emp.*, June, 1956, *63*, 385–389. (From: Department of Obstetrics and Gynaecology, University of Cape Town, Cape Town, South Africa.)

Two cases of labor in women with intrapelvic protrusion of the acetabula are reported. The "Otto" pelvis is defined as "an affection of the hip joint characterized anatomically by deepening of the acetabulum with mesial displacement of the inner wall." The cause or causes remain obscure, many disease states having been held as the responsible factors.

The condition is usually found in the middle-aged, but it has occurred even in young children and infants. Pain in the hip joint of thighs with limitations of all of the movements are the characteristic features. Roentgenologically the head of the femur is typically intact and buried deeply in the acetabulum. The medial and inferior walls of the acetabulum are thinned and lead to a dome-shaped protrusion into the pelvic cavity. The limitation of movement of the joint is the result of the head of the femur being deep within the acetabulum and the osteophytes, which often project outward at the junction of the head and neck.

Prolonged labor occurred in both of the cases presented. Fetal death ensued in one as a result of massive intracranial hemorrhage. In each a cesarean section should have been performed.—*John F. Weigen, M.D.*

Stevenson, Ralph Richards. (Washington, D. C.) Hysterosalpingofluoroscopy with simultaneous cine recordings. *Am. J. Obst. & Gynec.*, June, 1956, *71*, 1328–1334.

The author describes his results of both fluoroscopy and recorded moving picture studies with the image amplifier in hysterosalpingography. The value of multiple oblique studies of a hollow viscus has long been recognized. The author presents the first series of such studies done on the female generative tract.

The greatest technical disadvantage was due to the fact that no flexible cannula-tenaculum had been developed. However, the hinged polyethylene combination more recently discovered offers free movement, with less discomfort to the patient.

Teamwork between the radiologist and gynecologist is urged, in view of the radiation quality delivered within the pelvic area.

Two illustrative cases are presented with multiple frames from the recording. These show the advantages of oblique views and direct visually controlled rotation of the uterus by the tenaculum, held by the gynecologist.—*Eugene J. McDonald, M.D.*

Ahumada, Juan C., Nogués, Armando E., and Donovan, Cornelio. Hysterography in the diagnosis of dead and retained human ovum. *Am. J. Obst. & Gynec.*, June, 1956, *71*, 1274–1278. (From: Clinica Ginecologica de Buenos Aires, Buenos Aires, Argentina.)

Thirteen cases are presented to illustrate diagnoses of a dead fetus in early pregnancy made by hysterography.

The author states that hysterography should not be performed unless death of the embryo is certain. They present their positive findings which are: (1) The contrast medium acts upon an atonic wall enlarging the uterine cavity, thus producing an incomplete circular shadow, the boundaries of which are the uterine wall in the outer part and the fetal membranes in the inner part. The injection of the contrast medium is easily performed: at least 10 to 15 cc. of the medium should be used; and (2) the presence of the dead ovum in the uterus prevents the formation of a complete and uniform shadow.

In differential diagnosis they mention: (1) The typical image in the case of submucous myoma or interstitial myoma with submucous tendency, a most frequent error; (2) endometrial polyp; (3) adenomyosis; (4) endometrial hyperplasia; and (5) endometrial carcinoma.—*Eugene J. McDonald*, M.D.

Camiel, Mortimer R. Roentgen demonstration of placental calcification. *Radiology*, Aug., 1956, *67*, 218–223. (Address: 149 New York Ave., Brooklyn 16, N. Y.)

Roentgenographic demonstration of placental calcification has been reported and discussed by numerous authors. There is a wide difference of opinion as to its cause and its significance. Some have stated that its incidence is higher in patients who have received supplementary calcium and vitamins during their pregnancy; others believe calcification in the early months of pregnancy is physiologic, while that occurring late is of a degenerative nature. The author believes that it is a part of the complex biochemical changes related to placental aging. Whether normal or abnormal, it appears to have no adverse effect on the mother or fetus.

The author compared pre- and postparturient roentgenograms of calcified placentas in 5 cases in order to correlate the pattern of this calcification with that seen on pelvimetric films. These studies showed that the calcification occurs in a fine, lacelike, diffuse pattern—greater near the periphery of the individual cotyledon, and most marked at the placental rim, forming a wedge-like edge. The anatomic division into lobules and cotyledons could be easily identified. After study of the roentgenograms of the detached placentas, it was found that the same placental details could be identified in the preparturient films.

Placental calcification is of interest to the radiologist mainly because it confirms the location of the placenta on obstetrical films. Roentgen studies of the delivered placenta should also be of value to the anatomist and pathologist in examining placentas.—*Charles L. Stevenson*, M.D.

Genitourinary System

Cirla, Augusto, and Galdini, Salvatore. Contributo allo studio del rene a spugna. (Contribution to the study of the sponge kidney.) *Radiol. med.*, June, 1956, *42*, 605–610. (From: Istituto di Radiologia dell'Università, Milano and Istituto di Radiologia e Rep. Chirurgico dell'Ospedale S. Anna, Como, Italy.)

In recent years a number of papers have appeared in Italian and French radiologic literature dealing with segmental cystic dysplasia of the kidney which is usually referred to as sponge kidney.

The pathologic changes are as follows: 1) The kidney is increased in volume, slightly rounded in shape, but retains a normal surface and a normal color; 2) the cut surfaces show that the pyramids of Malpighi are very pale, contrasting with the normal color of the cortex, and that they present scattered, numerous small cysts. These cysts, which give the kidney a porotic appearance, may contain a serous liquid or small calcific deposits.

Microscopically, it appears that these changes are localized only in the pyramids of the kidney. The cysts are generally communicating with the intrarenal excretory pathways (which explains the findings on intravenous urography). Other cysts of varying sizes may be completely isolated and contain serous fluid.

The epithelial lining in both types of cysts does not show any evidence of inflammatory or degenerative changes.

It is generally thought that the sponge kidney is related to a dysembryoplastic condition, but while a few authors believe that this dysplasia is a pathologic entity in itself, others relate it to the polycystic kidney.

The clinical symptoms are not characteristic, and the diagnosis is made only after intravenous urography or, more seldom, on the scout film of the abdomen, when small, scattered calcific deposits are seen in the renal parenchyma. The pyelographic findings are characterized by the filling of small cavities symmetrically arranged around the calyces. The appearance may be compared to a bouquet of flowers, the stems forming the calyces and the flowers the cysts.

The previously described pathologic changes may be limited only to one kidney or to a segment of one kidney or even to one calyx.

In the differential diagnosis, pyelogenic cysts and nephrocalcinosis should be kept in mind.—*A. Govoni*, *M.D.*

RADIATION THERAPY

O'BRIEN, FREDERICK W., JR., and O'BRIEN, FREDERICK W. Irradiation and surgery in the management of invasive carcinoma of the cervix. *Radiology*, July, 1956, *67*, 1–9. (Address: F. W. O'Brien, Jr., 465 Beacon Street, Boston 15, Mass.)

It is the belief of the authors, after a thorough study of all the evidence, that radium and external irradiation constitute the most effective modalities in the management of invasive carcinoma of the cervix. They feel that the clinical radiotherapist must individualize the treatment in each patient. They use radium distributed, so as to deliver a tumor-lethal dose to the cervix, endocervix, cervical canal, vaginal apex, paracervical triangle, and the adjacent parametrium with the latter augmented by external irradiation. The use of a simple method of applying this gives more uniform results. The Clark or inverted T applicator and the Ernst device are preferred. One hundred milligrams of radium are used in the Clark applicator with 20 mg. in the uterine canal, 30 mg. in the endocervical section and 50 mg. in the cross bar. The bladder and the rectum are carefully packed away and no indwelling catheter is used. Dosage is calculated after anteroposterior and lateral roentgenograms of the pelvis are obtained and after an air-filled Foley catheter is placed in the bladder and barium instilled in the rectum. In using the Ernst applicator, only one application is made as it is easier and more economical for the patient and since it has produced no undesirable effects.

Roentgen therapy is employed to complement the radium dose in the parametria. The authors prefer to start this about three weeks after the radium application. In Stages I and II cases, as a rule, 4 and sometimes 6 portals are used, depending on the size of the patient, and about ten weeks are required to deliver the desired depth doses. In Stages III and IV cases, transvaginal irradiation with supplementary external irradiation is preferred. Complications such as transient diarrhea and tenesmus and bladder symptoms may occur, but these are usually amenable to supportive measures. The point is made that the complications of fistulas, ureteral obstruction, and necrosis are due to the disease itself, rather than to results of irradiation.

Interest in the surgical treatment of carcinoma of the cervix was revived by the publication of Meigs' paper in 1947, in which he reported 91 operations of the Wertheim-Taussig type with no mortality. His reported results were as good as, and even slightly better, than those obtained by combined treatment with radium or roentgen therapy and surgery. The Wertheim-Taussig operation is a formidable procedure—one not to be used by the "occasional hysterectomist," but only by those who carefully select and prepare their patients and can provide the ideal preoperative and postoperative care and follow-up. A recent report by Liu and Meigs covers 193 patients with primary invasive carcinoma of the cervix who had been operated upon and followed for five years or more. In this series there is no conclusive evidence that surgery is superior to irradiation.

A cytologic test developed by the Grahams is discussed. This vaginal smear method proposes to show a correlation between basal cells, the sensitization response, and positive lymph nodes. The work of Glücksmann is a similar effort to develop a method by which the effects of radiation on malignant cells *in vivo* can be measured quantitatively.

The follow-up of the patient is important and should consist of a complete pelvic examination, Papanicolaou smears, biopsy and other diagnostic procedures on a monthly basis. The treatment of recurrences must be carefully planned and managed by a pathologist, cytologist, cancer surgeon and the radiotherapist. Radium therapy may be repeated, but must be considered as a calculated risk, and surgical intervention may be possible in selected cases.—*Donald N. Dysart, M.D.*

RANDALL, JOHN H., and GODDARD, WILLIAM B. A study of 531 cases of endometrial carcinoma. *Surg., Gynec. & Obst.*, Aug., 1956, *103*, 221–226. (From: Department of Obstetrics and Gynecology, State University of Iowa Hospitals, Iowa City, Iowa.)

The authors present a comprehensive analysis of 531 cases of endometrial carcinoma which were seen in the Department of Obstetrics and Gynecology of the State University of Iowa Hospitals during the period from January, 1926 to January, 1951. Of these, 516 received treatment with an over-all five year survival rate of 55.2 per cent. Eighty per cent of the patients were postmenopausal and the greatest incidence occurred between the ages of fifty to fifty-nine years. Roughly, one fourth had never been pregnant and about one half of them weighed 170 pounds or more at the time of examination. Fifty-eight per cent were considered to have had a late menopause, whereas twenty-nine patients had had an artificial menopause, 22 by irradiation and 7 by oophorectomy.

Although various combinations of radium therapy and surgery were employed throughout the twenty-five year period, the preferred plan was intracavitary radium followed in six to eight weeks by total hysterectomy. The use of multiple radium foci was found superior to the use of an intrauterine tandem. With this combination of methods, a five year survival of 75.77 per cent was realized, whereas the survival with radium alone was 49.29 per cent and with surgery alone was 60 per cent. Examination of uteri removed following radium therapy disclosed that tumor invading the myometrium was apparently more difficult to destroy. In the instances

where residual tumor was found, the survival rate was 61.3 per cent, in contrast to a salvage of 76.3 per cent when the tumor had been sterilized. Prognosis could be definitely related to stage of the disease, the best results being obtained in low grade tumors entirely confined to the uterus.

Coexisting diseases seemed to have little direct effect upon the outcome. Uterine fibroids present in 22.8 per cent of the series produced enough irregularity of the uterine cavity in some cases to make currettage difficult and require some modification in the plan of therapy. The most common generalized disease was cardiovascular disease (43.7 per cent) which in combination with hypertension and cardiorenal complications materially affected operability and longevity. The operative mortality accompanying hysterectomy was 3.2 per cent with no deaths occurring after 1946.—*James A. Martin, M.D.*

Batten, Richard, and Brown, D. E. Meredith. Protection of ovaries from radiation. *Lancet*, June, 1956, *1*, 939–940. (From: Hospital for Sick Children, Great Ormond Street, London, England.)

The authors present an unusual case history of an eight year old girl who was found to have a large pelvic retroperitoneal neuroblastoma. At laparotomy, this diagnosis was confirmed and both ovaries were ensheathed in lead shields covered with rubber finger cots and attached to the anterior parietal peritoneum as high as possible. Then radiation therapy was given over the pelvis with 250 kv., a total depth dose of 2,150 r being delivered in thirty days. At a second laparotomy the shielding was removed from both ovaries and a pericolic abscess which had formed on one side was drained.

Calculations revealed that the right ovary had probably received about 50 r, while the left ovary which was in the field of direct radiation received about 170 r.

Three years later the patient was in good general health without evidence of recurrent or metastatic tumor. Physical development was proceeding naturally, and menstruation which had begun six months previously was occurring with regular twenty-eight day cycles.—*James A. Martin, M.D.*

Lindell, Bo, and Walstam, Rune. A new telegamma apparatus. *Acta radiol.*, March, 1956, *45*, 236–248. (From: Institute of Radiophysics and Radiumhemmet, Karolinska Sjukhuset, Stockholm, Sweden.)

This article is a description of a new cobalt 60 unit which is a modification of the Radiumhemmet radium unit but which permits more variety of setups and shorter treatment times. It is ceiling suspended and contains a rotating lead drum which gives at least 12 cm. of lead protection to the 8.5 curie source.

The source is moved into the tubular treatment head by rotating the channeled drum and pushing it with a wire. It has the shape of a cylinder 6 mm. long and 6 mm. in diameter and is considered a point source, permitting taking-off of the beam from two different places on the treatment head. This, together with the use of diaphragms permits multiple types of applications.

Distances used vary from 6 to 10 cm. skin-source distance. The outputs were of the order of 55 r per minute at the short skin-source distance to 20 r per minute at the long source-skin distance.

Depth doses are shown and compared with those of teleradium therapy. In general, with cobalt 60 this equipment gives a more uniform dose and a sharper edge to the treatment field than the teleradium unit. No clinical data are given but apparently the unit is used primarily for multiple field therapy of lesions about the head and neck.—*Henry J. Klos, M.D.*

RADIOISOTOPES

Pahaut, Jean E., and Govaerts, Jean. Utilisation du radiostrontium ^{89}Sr dans le traitement du cancer. (Use of radioactive strontium Sr^{89} in treatment of cancer.) *J. de radiol. et d'électrol. & Arch. d'électric. méd.*, March-April, 1956, *37*, 164–169. (From: Centre de Radiologie de la Clinique Saint-Joseph et Institut de Physique Nucléaire de l'Université de Liège, Belgium.)

The authors present 3 cases of bone tumors treated by a combination of external roentgen therapy and radioactive strontium (Sr^{89}). Sr^{89} has a half life of fifty-five days and is essentially a pure beta emitter with a maximum energy of 1.3 mev.

A total of 30 gm. of $Sr^{89}CO_3$ is given orally at the rate of 6 tablets a day for five days.

Urine and feces are saved and counted for a period of ten days. Blood samples are taken every day for ten days and blood tests are made frequently.

In treatment of different kinds of bone tumors it was observed that Sr^{89} was selectively deposited in the newly formed bony tissues.

Concentration factors were calculated to be of 12 per cent in a case of Ewing's sarcoma, 7 per cent in a case of osteoblastic metastases and of 4 per cent in a case of chondrosarcoma.

The evaluation of the dissipated energy calculated in rep or in gram rad shows that the effect of the beta emission is remarkable.

There was no systemic damage observed by administration of adequate dosage and the authors feel that the use of Sr^{89} in combination with external roentgen therapy in certain cases of fast growing bone tumors may improve considerably the therapeutic results.—*Maurice Dufresne, M.D.*

COCKCROFT, SIR JOHN. The future role of atomic energy in industry, science and medicine. *Brit .J. Radiol.*, May, 1956, *29*, 256–260. (From: Atomic Energy Research Establishment, Harwell, England.)

1. *Application to Industry—Nuclear Power.* The most important application of atomic energy to industry will come from the generation of nuclear power. Twelve nuclear power stations are scheduled for completion by 1965. It is predicted that by 1975 about 40 per cent of the electricity will be produced from this source. Fourteen tons of fission products will be produced. Radiocesium and radiostrontium are to be separated from the fission products.

2. *Application of Radioactive Isotopes in Industry.* A) Radiography by gamma rays is widely used for inspection of wells, castings, forgings, and assemblies. Cobalt 60, iridium 191, cesium 137 and thulium 170 are used; B) Noncontact thickness gages using absorption or backscattering to measure continuously the thickness of materials; C) Detection of corrosion of pipes by backscattering means; D) Detection of leaks in water pipes using weak radiosodium in solution; E) As a polymerisation agent; F) Sterilization of pharmaceuticals, sutures and dressings and G) Preservation of food stuffs.

3. *Application of Radiation and Radioisotopes to Medicine.* The use of cobalt 60, cesium 137, iodine 131, gold 198, and sodium 24, for radiation therapy is well known. The treatment of brain tumors by injecting boron 10 into the tumor and treating the tumor with neutrons to produce alpha particles has been done. Thulium 170 and xenon 133 have been used for diagnostic radiography. Iodine 131 is being used to study thyroid functions and disorders and iron 59 to study diseases of the bone marrow. Cardiac output can be measured by injecting serum albumin labeled with iodine 131 or chromium 51 into the blood stream. Plasma volume can be determined. Total water volume can be determined with the aid of tritium. Brain tumors con be located by injecting iodine 131 di-iodo-fluorescein.

4. *Application of Atomic Energy to Science.* Biologic research is the most important application. Dr. MacFarlane found by carbon 14 labeled compounds that practically every tissue in the body is in continuous exhange and that in the animal relatively few atoms and molecules that are present in youth are present in old age. Amino acids have been found to be assembled into proteins in 25 minutes. The metabolism of steroid hormones has been investigated. Photosynthesis has been studied. Radioactive phosphorus 32 and calcium 45 have been used to help understand the factors influencing soil fertility. Radiation can be used to speed up mutations in plants and thus perhaps obtain a better product.

The application to physical science includes the production of elements of atomic nuclei 93 to 101 by successive neutron capture in atomic piles coupled with transmutation by heavy particles. Transistors have been improved by neutron bombardment.—*Charles W. Cooley, M.D.*

THOMAS, COLIN G., JR. Lymphatic dissemination of radiogold in the presence of lymph node metastases. *Surg., Gynec. & Obst.*, July, 1956, *103*, 51–56. (From: Department of Surgery, School of Medicine, University of North Carolina, Chapel Hill, North Carolina.)

In order to study the distribution and effects of colloidal radioactive gold in lymph nodes, this pilot study was carried out.

Doses of from 0.1 to 5 millicuries in a 1 cc. volume were injected into large, or near small tumors, usually from 20 to 24 hours before surgery. After surgery, the location of all identifiable lymph nodes was plotted and they were assayed for activity with a collimated Geiger-Müller tube and examined histologically for the presence of lymphoid and tumor elements. Darkfield examination revealed the location of the radioactive gold.

Thirty five patients were studied; 23 with breast carcinoma and 12 with head and neck cancer. Of the total of 593 lymph nodes examined, 374 contained radiogold. Of the 94 cancerous lymph nodes, only 33 contained the radiogold.

There seemed to be a uniform distribution of the colloidal radiogold through the lymph nodes if there was no metastatic involvement, some of the material apparently passing through the proximal lymph nodes without delay. In the presence of cancer metastasis to lymph nodes, the distribution of the radiogold was spotty and most cancerous lymph nodes contained no detectable radioactivity.

The author concludes that treatment of metastatic cancer by lymphatic dissemination of radioactive gold is unlikely to succeed and that attempts at such therapy are hazardous.—*Henry J. Klos, M.D.*

BONTE, FREDERICK J., STORAASLI, JOHN P., and WEISBERGER, AUSTIN S. Comparative evaluation of radioactive colloidal gold and nitrogen mustard in the treatment of serous effusions of neoplastic origin. *Radiology*, July, 1956, *67*, 63–66. (Address: F. J. Bonte, 5203 Harry Hines Blvd., Dallas, Texas.)

The authors present a comparative evaluation of radioactive colloidal gold and nitrogen mustard in the treatment of serous effusions of neoplastic origin.

Since its introduction by Müller in 1949, intracavitary radioactive colloidal gold therapy for carcinomatous effusions has become an almost universally accepted mode of treatment. Those who have worked with colloidal Au^{198} realize, however, that there are certain disadvantages inherent in its use. Since several authors have called attention to nitro-

gen mustard as an agent which is also effective in the control of malignant effusions, and as some of the disadvantages of treatment with Au^{198} are not present in HN_2 therapy, a comparison of the results obtainable by these two methods of treatment seemed in order.

It is recommended that interested readers review the original article for a detailed account of the methods used in both forms of therapy during this clinical study. However, in brief, the method followed in the administration of radioactive colloidal gold was the same as that used in most institutions at this time. Treatment with nitrogen mustard consisted of the administration of a single injection of 0.4 mg. per kilogram of body weight, in 50 to 100 cc. of sterile normal saline. It is pointed out that a free flow of pleural or peritoneal fluid must be present before instillation of NH_2, in order that sufficient remain to insure dissemination of the mustard and to guard against the introduction of the substance subcutaneously or into a loculated pocket. For pericardial effusion, a dose of 0.2 mg. per kilogram of body weight was generally employed.

A total of 60 patients were treated with Au^{198}, 40 patients with HN_2, and 12 with both medications. It was found that HN_2 directly instilled into malignant effusions appears to be of the same order of effectiveness as radioactive colloidal gold. Mustard, however, was noted to have several advantages over radioactive gold: (a) it is easier and simpler to administer, and no special facilities are required for its use; (b) it is almost universally available; (c) it is less expensive to the patient; and (d) it presents no radiation hazard either to the nursing personnel or to those administrating the treatment. The study also showed that if nitrogen mustard proved ineffective, subsequent treatment with Au^{198} was often beneficial. The authors did not believe they had proven, however, that combined form of therapy, involving the use of both materials, gave better results than could be obtained with either used alone. —*A. Ralph Watson, M.D.*

Siegel, Elsie P., Hart, Hiram E., Brothers, Milton, Spencer, Herta, and Laszlo, Daniel. Radioyttrium (Y^{90}) for the palliative treatment of effusions due to malignancy. *J.A.M.A.*, June 9, 1956, *161*, 499–503. (Address: D. Laszlo, 210th Street and Bainbridge Avenue, New York, N. Y.)

The intracavitary administration of radioyttrium (Y^{90}) in the presence of effusions due to malignancies results in good localization of Y^{90} on serosal surfaces. This is thought to occur by combination of Y^{90} with the constituents of malignant effusions, mainly proteins, resulting in colloidal formations of radioyttrium. As compared with other radioisotopes used in a similar manner, advantages of Y^{90} in the palliative treatment of effusions due to malignancies include: it is a pure beta emitter, it has a relatively short half life of 61 hours, and a specific agent is available to rapidly remove it from the circulation if it is inadvertently introduced into a vascular channel.

The preparation of solutions of Y^{90} and methods of administration are described in some detail. Surface dose in rads in a hypothetical case of pleural effusion is given and marked fall off in dose at depth, characteristic of beta emitting isotopes, is illustrated. Studies of the distribution are included and demonstrate clinically insignificant levels in blood, urine, liver and bone during intracavitary therapy.

Sixteen cases receiving radioyttrium are presented, 13 intrapleurally and 3 intraperitoneally. Results in 8 cases were considered good and clinical and laboratory studies demonstrated no untoward side effects during or following treatment. Autopsies on 4 cases revealed varying degrees of fibrosis of treated serosal surfaces and a normal appearance of adjacent viscera and bone marrow.

The authors conclude that because of the favorable radiation characteristics of Y^{90} and the resultant increased safety to patients and personnel, radioyttrium may well deserve a place in the radiotherapist's armamentarium.—*John T. McLelland, M.D.*

Osborne, J. C., and Kowalewski, K. The uptake of radiosulfur in the fractured humerus in the rat. *Surg., Gynec. & Obst.*, July, 1956, *103*, 38–40. (From: McEachern Cancer Research Laboratory and the Department of Surgery, University of Alberta, Edmonton, Alberta, Canada.)

It has been shown that radiosulfur (S^{35}) injected into the rat can be demonstrated in the long bones within a few hours, particularly in the cartilage of the epiphyses. It is known that cartilage formation is a phase in the repair of fractures.

Watson-Jones has stated that if a fracture is not immobilized, there is an increase in the amount of cartilage formed during healing. This study was undertaken to test that hypothesis.

It was found that at three weeks the amount of radioactive sulfur in the fractures of unsplinted rat humeri was greater than that in splinted humeri, tending to support Watson-Jones' statement. No histologic studies were performed to confirm this, the assumption being that the increased amount of sulfur reflected the presence of increased cartilage.

The sulfur was precipitated out as barium sulfate following digestion of the organic material of the bone and then counted with the Geiger-Müller tube.—*Henry J. Klos, M.D.*

Dudley, H. C., Markowitz, H. A., and Mitchell, T. G. Studies of the localization of radioactive gallium (Ga^{72}) in bone lesions.

J. Bone & Joint Surg., June, 1956, *38-A*, 627–637. (From: United States Naval Hospital, St. Albans, New York, N. Y.)

It has been shown that radioactive gallium (Ga^{72}) when administered as a citrate is deposited in bone and has a particular affinity for areas of osteoblastic activity. The concentration gradient between normal muscle and bone is of the order of 1 to 50, while the ratio between normal and proliferating bone is approximately 1 to 4. Following very detailed description of the preparation and standardization of Ga^{72} and counting techniques used in their laboratories, the authors describe their procedure of administration, method of scanning and results in a series of 66 proved cases of malignant bone lesions and 49 cases of nonmalignant bone lesions.

In the authors' experience, intravenous administration of 2 millicuries of Ga^{72} followed after 48 hours by a scanning procedure utilizing a scintillation counter proved most effective. At this time the differential concentration between Ga^{72} in abnormal bone and adjacent tissue was at a maximum. A consideration of false positive and negative findings closely related apparent discrepancies in Ga^{72} concentration to regional blood supply. In the 66 malignant lesions, stated results seemed to indicate that the present scanning technique of the authors has limited usefulness as a routine diagnostic procedure. It may serve some purpose in studying the extent of a malignant lesion involving bone, and perhaps in studying the response of malignant lesions of bone to therapy. In the study of nonmalignant lesions information obtained relative to bone infection was inconclusive because of the small number of patients studied but a correlation between stages in bone healing and Ga^{72} concentration in fractures was noted and suggested that Ga^{72} tracer study techniques may deserve a place in the study of metabolism of healing bone lesions.—*John T. McLelland, M.D.*

MAYNEORD, W. V., and IRELAND, H. J. D. Radiography with thulium 170 and xenon 133. *Brit. J. Radiol.*, May, 1956, *29*, 277–285. (From: Department of Physics, Institute of Cancer Research, Royal Cancer Hospital, London, S.W.3, England.)

Radioactive isotopes emitting low energy x-rays may have a place in human radiography as well as in certain industrial applications. Strong thulium sources have now, however, been shown to emit intense high energy bremsstrahlung. The authors have therefore investigated the possibilities of other radioactive isotopes, particularly Xe^{133}.

A source holder designed for clinical use is described in detail, including internal applications.

It is important to know the approximate dose rates in the vicinity of the source, both from the point of view of estimating necessary radiographic exposures and that of protection. Measurements were therefore made with air wall condenser chambers calibrated in roentgens. The maximum permissible dose rate of 300 mr/week (approximately 2 mr/h. for continuous irradiation) is reached at a distance of approximately 70 cm. This dose rate is found experimentally to be reduced by a factor of 0.28 by an additional thickness of 3 mm. of lead. The maximum permissible level is then reached at a distance of 37 cm.

It was found that satisfactory negatives could be produced with an exposure time of 20 sec. at a source-film (focal) distance of 10 cm.

The effect of a moving Bucky diaphragm was investigated. It was found that some slight improvement was obtained in the definition, at the expense of a 2 to 1 increase in exposure time.

The main difficulty in general clinical application is still the comparatively long exposure time required, though this has been reduced from many hours to a few seconds. The need is obviously for still stronger sources. If such sources are obtained by the use of larger masses of thulium, then the penetrating bremsstrahlung becomes of greater importance and loss of contrast results.

The presence of the bremsstrahlung from the thulium source has two undesirable effects (a) the loss of radiographic contrast, and (b) increased difficulty in protection.

Xenon 133: Xenon 133 might prove to be a suitable radiographic source and, again, the problem is essentially one of availability in sufficient strength and specific activity. Xe^{133} is present as a high yield (6 per cent) fission product in large quantities of reactors of even moderate power. The problems of chemical and physical separation exist, rather than those of neutron activation.

The design of a suitable source is dominated by the necessity for virtually a point source. This, of course, requires that an extremely high specific activity be attained, which normally means that the gas would have to be highly compressed into a small volume.

The source consists of a tube 3 mm. in diameter and about 2 cm. long, flattened and having the radiographic source near one end. This tube may then be inserted into a protective housing and suitable shutter mechanism. Owing to the fragile nature of the aluminum container, sealed glass capillary tubes are preferable for very strong sources.

A large number of radiographs have been taken under various conditions. When these are compared with similar pictures for Tm^{170} it becomes clear that the radiographic contrast is better in the case of Xe^{133}, probably owing to the absence of high-energy radiation.

For the normal ankle the source to film distance was 30 cm., the dose rate being about 13 mr/min. and the exposure time 40 seconds.

The maximum permissible body burden of xenon 133 is 320 μc and the maximum permissible concentration in air $4\times10^{-6}\mu$c/cc. It is an inert material whose metabolism may be regarded as similar to that of other rare gases such as krypton.

In practice, now, two types of source are required; one for large focal distances and therefore of high radioactive content, and a second of lower content but small size, for use at short focal distances such as occur in dental radiography with the source in the mouth.

In circumstances in which its short half-life is not a serious disadvantage Xe^{133} appears to give more satisfactory radiographs than Tm^{170} and is less hazardous in use.

For field work in time of war and for accidents such an x-ray unit has obvious advantages. By combining this highly portable x-ray apparatus with a rapid developing process which requires no darkroom (Picker-Polaroid process) the maximum possibilities of the method may be obtained.—*Stephen N. Tager, M.D*

MISCELLANEOUS

Case, James T. Fifth Inter-American Congress of Radiology: Opening address. *Radiology*, April, 1956, *66*, 599–601. (Address: 2315 Bath St., Santa Barbara, Calif.)

In his opening address to the Fifth Inter-American Congress of Radiology, Dr. Case traced the development of this Congress from the first meeting of radiologists of all the Americas in 1933, as The American Congress of Radiology through the first Inter-American Congress of Radiology in Buenos Aires in 1943 and subsequent meetings in Havana, Santiago, and Mexico City. The Inter-American Congresses are held under the auspices of the Inter-American College of Radiology which developed from the meeting in Buenos Aires. The admirable contributions to medical radiology of many Latin American radiologists were recalled, as well as the outstanding educators who have represented Latin American radiology.

Dr. Case called attention to the responsibilities and opportunities resulting from the development of atomic energy and the need for keeping thoroughly informed on the new developments. The expanding peacetime applications of atomic energy will greatly increase radiation hazards, placing additional responsibility on physicians, and on radiologists especially.—*Harold C. Hamilton, M.D.*

Marquès, P., Bru, A., and Delpha M. Dosimétrie en plésioroentgentherapie (tube Philips Métalix). (Dosimetry in contact roentgen therapy: Philips Metalix tube.) *J. de radiol. et d'électrol. & Arch. d'électric. méd.*, March-April, 1956, *37*, 170–172. (From: Centre anticancéreux, Toulouse, France.)

Measurement of isodose curves with the Philips Metalix apparatus operating at 50 kv. can be obtained accurately with a Sievert micro chamber.

The authors report the results of studies undertaken to evaluate differences in the dose distribution, employing in one case, a horizontal or flat cone as port of entrance and in the other case, a spherical type of cone depressing the skin.

By comparison they found that when depressing the skin with the spherical type of cone, a much more homogeneous surface dose could be obtained and the authors finally suggest that in contact therapy whenever possible this kind of technique should be employed for better therapeutic results.—*Maurice Dufresne, M.D.*

Morgan, J. E., and Ellinger, F. Radiation dosimetry in biological research. *Radiology*, June, 1956, *66*, 877–886. (Address: J. E. Morgan, Naval Medical Research Institute, National Naval Medical Center, Bethesda, Md.)

An impression has prevailed among many physicists that biologic research data are necessarily less accurate than physical data. This has been thought natural since the lack of uniformity among living experimental subjects would preclude a satisfactory reproducibility of results while the rigidly controllable inanimate material would permit this reproducibility. Modern statistical methods, however, together with painstaking care in the selection and handling of animals now permit an order of accuracy in radiobiologic experiments that approach the precision attainable by physical dosimetry.

The authors discuss in this paper methods which they have found useful in minimizing some of the more obvious sources of dosimetric uncertainty. Three major types of roentgen-ray generators are currently being employed for animal irradiation experiments in the Naval Medical Research Institute. These consist of: a Picker 220 kvp. therapy unit, a General Electric 250 kvp. Maxitron, and a 2 mev. Van de Graaff accelerator. A cobalt irradiator which consists essentially of 60 individual 25 to 50 curie sources is being made use of and a detailed report of its design and performance characteristics will be made later.

The Picker 220 kvp. unit has been used for irradiation of small animals at voltages between 140 and 200 kvp. Since variations in line voltage will vary the output intensity, a 5 kva. stabilizer in the incoming power line was installed and this reduced line fluctuations from $\pm$10 per cent to around 0.5 per cent or less. Fine kv. adjustments, not obtainable with the autotransformer control unit on the original machine, were made available by installing a Variac which provides smooth vernier action between steps of the regular potential selectors. A thermal drift encountered in the panel meters which showed a

steadily increasing deviation between the meters, was eliminated by hanging an aluminum baffle sheet between the rheostat coils and nearby kilovolt meter and installing a large centrifugal blower in one of the two existing cooling apertures at the top of the control cabinet.

Unstable line voltages were remedied on the 250 kvp. Maxitron unit by use of a thyratron-controlled, motor-driven Variac, which superposes a "buck or boost" voltage on the incoming line as needed to compensate for these fluctuations. Since the kilovolt indicator on this unit tended to be rather sluggish in response, too coarsely calibrated for optimum precision, and subject to some thermal drift during long exposures a precision voltmeter was permanently connected across the high-tension primary and this is being used as the principal standard of reference.

Very accurate control of the accelerating potential is necessary when using the 2 mev. Van de Graaff generator. Variables in addition to those above consist of driving motor speed, belt slippage and charging efficiency, corona and leakage current losses. These factors necessitated the use of a device capable of accurately indicating some quantity linearly related to the radiation output so an ionization chamber fixed in the tube head and directly connected to a galvanometer circuit was installed and provided a reference point for all control adjustments.

The Victoreen condenser r meter is used for dose measurements below 200 to 300 kv. Above these levels a continuous reading galvanometer circuit, precalibrated against the condenser chambers at moderate roentgen-ray voltages is employed. A special type of miniature ionization chamber, found most useful for depth and isodose measurements, is described. An extrapolation technique for obtaining accurate timing readings is discussed and illustrated.

All of these factors permit the measurement and delivery of precisely standardized doses of roentgen and gamma radiation so essential for radiobiologic laboratory experiments.—*Donald N. Dysart, M.D.*

V. D. DECKEN, CLAUS-BENEDICT. Plexiglas als Phantommasse für die Messung von Dosisverteilungen schneller Elektronen. (Plexiglass as a phantom material for the measurement of dose distribution of fast electrons.) *Strahlentherapie*, Feb., 1956, *99*, 227–229. (From: Czerny-Krankenhaus für Strahlenbehandlung der Universität, Heidelberg, Germany.)

With fast electrons of 15 mev. energy in a beam 10 cm. in diameter, actual measurement with an ionization chamber of doses to depths of 7 and 8 cm. in water and in plexiglass showed the plexiglass to be water equivalent for all practical purposes.—*Henry G. Moehring, M.D.*

WILLIAMS, WILLIAM L., STONER, RICHARD D., and HALE, WILLIAM M. A correlation of early radiation changes in lymphatic tissues with antitoxin-producing ability. *Yale J. Biol. & Med.*, June, 1956, *28*, 615–621. (From: Division of Pathology and Microbiology, University of Tennessee, Memphis, and Division of Microbiology, Department of Medicine, Brookhaven National Laboratory, Upton, Long Island, N. Y.)

The authors report on the correlation of the morphologic changes in lymphatic tissues of mice following irradiation with the ability of the animal to produce tetanus antitoxin, and also correlate the changes in morphology of the thymus of old mice with the increase in the ability of thymic transplants from old mice to produce antitoxin.

The metabolic changes which influence the production of antibodies occur following the first six hours after irradiation. The mature lymphocytes and plasma cells that have been said to be the sites of antibody production have usually undergone complete necrosis within this period. The early changes reflecting the interference with antitoxin production are occurring in the cells of the phagocytic or reticuloendothelial system. This is also reflected in studies of age involution in the thymus where it is seen that the absence of mature cells does not interfere with antitoxin production.

The authors believe they have demonstrated the ability of undifferentiated cells of the reticuloendothelial system to produce antibodies in the absence of mature lymphocytes or plasma cells. Their concept of these cells is that while they are members of the reticuloendothelial system most of them have the ability to phagocytize under the proper stimulus. They apparently represent a multipotential cell which may differentiate into a transitional cell and thence into a lymphocyte, plasma cell or phagocytic cell under the proper stimulus.—*Edgar C. Davis, M.D.*